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THIOCYANATE THERAPY OF HYPERTENSION

STUDIES ON THE CONSTANCY OF BLOOD CONCENTRATION AND URINARY OUTPUT

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THIOCYANATE was originally studied pharmacologically by Bernard,[1] in 1857, but it was first applied as a therapeutic agent by Nichols,[2] in 1925. There then followed a group of sanguine reports of its efficacy as an agent in the control of high blood pressure, as exemplified by the studies of Gager,[4] Smith and Rudolf,[5] Bolotin,[6] Maguire,[7] and Palmer, Silver, and White.[8]

This buoyant period was followed by a series of reports of toxic effects by such writers as Ayman,[9] Palmer and Sprague,[10] and Goldring and Chasis.[11] With the introduction, in 1936, by Barker,[3] of a method of controlling the dosage of the drug by means of blood concentration estimations, we have arrived at a sane, middle-of-the-road policy. We are now better able to evaluate the drug, ascertain its efficiency, and control the dosage. That toxicity was caused by lack of control of the dosage is evidenced by the reports of O'Hare and his associates.[12] In 1931, before the introduction of Barker's method for ascertaining the blood level of the thiocyanates, and using empirical doses, these authors reported favorable results in only two out of twenty-five cases. Moreover, "every patient complained of one or more distressing symptoms arising from two to eight days after the drug was commenced." A study of the amount of thiocyanate used in their cases in the light of present knowledge readily explains the rapid onset of toxic symptoms. They gave their patients 1 Gm. daily for one week, then 0.6 Gm. daily for one week, and, finally, 0.3 Gm. daily. In the majority of patients such an early heroic dose will yield blood levels of 20 mg. per 100 c.c. or more, and naturally will bring them, in two to eight days, to a period

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of toxicity. As we shall demonstrate, as little as 0.1 Gm. daily may be sufficient in some cases to maintain a therapeutic blood level.

In 1938, using Barker's method of control, O'Hare and his associates[13] again tried the drug on fifteen patients. This time they reported that "in all cases a definite lowering of the blood pressure occurred during the interval of treatment." They found that the symptoms returned when the drug was surreptitiously omitted, and concluded: "the regulation of administration required the individualization of dosage for each patient, frequent blood cyanate determinations, and sufficient intelligence on the part of the patient to permit an awareness of early toxic symptoms."

The truth, as would be expected, lay in the fact that a very important element in the therapeusis had been overlooked; namely, failure to evaluate the results on the basis of the amount of drug available for action in the circulating blood, rather than the dosage prescribed. This fact was clarified by the work of Barker in his original report in 1936[3] and in his subsequent writings.[14-16, 26] Many other workers[17-24] have confirmed his results, so that now it is pretty well conceded that no therapeutic effect with thiocyanate can be truthfully judged without adequate blood level determinations at fairly frequent intervals. A study of the unfavorable reports reveals that the toxic manifestations and fatalities were most probably caused by excessive dosage. These unfortunate sequelae may very well have been averted by a proper determination of the blood level, for the dosage, per se, is no indication of the amount of the drug available in the circulating blood. On the contrary, the full therapeutic effect may be obtained with a very small dose in one case, whereas many times the same amount of drug may be required for another.

CLINICAL RESULTS

We have adequately followed thirty-nine ambulatory patients in our clinic during the past three years. The patients had essential hypertension, and many of them showed some evidence of cardiac or renal complication. There were seventeen males and twenty-two females, and their ages ranged from 34 to 71 years; the average age was 55 years.

The method employed was as follows: All of these patients had been observed for months, and many for years, with the usual therapeutic agents, so that the base level of their blood pressures was known. After the period of observation, potassium thiocyanate therapy was instituted. We used an aqueous solution of the drug in such concentration that one dram contained 0.1 Gm., and commenced with a dose of one dram three times daily. After one week the patient was examined, the blood pressure recorded, and a blood specimen was obtained. All blood pressures were taken in the recumbent posture, and after the patient had rested for at least ten minutes in that position. Many of the observations were checked by more than one physician. Since ours is an ambulatory cardiac clinic, held only once weekly, and the thiocyanate determination could not be performed immediately, the dose was maintained and the

patient was told to report the following week, at which time the dose was adjusted according to the blood level. Although in most patients the average blood level with this dose (0.1 Gm. three times daily) was 5 mg. per 100 c.c., as high a level as 15 mg. per 100 c.c. was obtained with this small amount. In those cases in which there were signs of congestive heart failure, digitalis therapy and diuretics were continued. Such a regime was not incompatible with the thiocyanate.

The following five cases were selected as typical examples of both the favorable and unfavorable results:

CASE 1.—(Fig. 1.) M. K., a woman, aged 48 years, was first admitted to the cardiac clinic May 31, 1938, with a diagnosis of (a) hypertension, (b) enlarged heart, (c) regular sinus rhythm, (d) Class III C. Her chief complaints were dyspnea, cough, palpitation, precordial pain, edema of the ankles, fatigue, pallor, and cyanosis. The blood pressure on admission was 240/130. A teleoroentgenogram revealed that the heart was enlarged in all diameters. There were straightening of the left cardiac border and accentuation of the right auricular curve. The aorta was not dilated. In addition, there was roentgenologic evidence of fibroid changes at the roots and bases of both lungs, especially on the right side. There was no pleural effusion. The electrocardiogram revealed a rapid sinus rhythm, with deviation of the electrical axis to the right. Numerous urine examinations showed a good ability to concentrate; the specific gravity ranged from 1.002 to 1.027, and there were no abnormal elements. The nonprotein nitrogen of the blood was 35 mg. per 100 c.c., and the glucose, 84 mg. per 100 c.c. The hemoglobin was 98 per cent, the erythrocyte count, 4 million per cu. mm., and the leucocyte count, 11,700 per cu. mm., with 70 per cent polymorphonuclear cells, 24 per cent lymphocytes, and 6 per cent transitionals. The blood Wassermann reaction was negative.

She was treated with phenobarbital, digitalis as indicated, and numerous expectorant mixtures for the chronic cough. During this period she showed persistent hypertension, with the systolic pressure between 190 and 240, and the diastolic between 116 and 140.

Potassium thiocyanate therapy was instituted after an observation period of ten months. The initial dose was 0.1 Gm. three times daily. One week later the blood pressure was still elevated, there was no change in her symptoms, and a blood thiocyanate determination showed 3.6 mg. per 100 c.c. The dose was increased to 0.2 Gm. three times daily, and, a week later, the blood pressure had fallen to 172/104 and the blood thiocyanate was at a level of 13.3 mg. per 100 c.c. There were no toxic symptoms, and the patient felt considerably improved, so the dose was continued at 0.2 Gm. three times a day. The next week she began to have dizziness; the blood pressure had continued to fall, reaching a level of 166/114, and the thiocyanate concentration had risen to 18.2 mg. per 100 c.c., so the dose of potassium thiocyanate was reduced to 0.2 Gm. twice daily. Shortly after this the patient was hospitalized for an acute exacerbation of her chronic cough, and did not take any thiocyanate for a period of three weeks. On her return to the clinic, the blood pressure was 194/114, and the dose of 0.2 Gm. twice daily was resumed. She has since been maintained on the thiocyanate treatment; the dosage has been determined by the blood level, except for an occasional period when she has not returned to the clinic for care. After these latter periods, a resumption of the high level of the blood pressure occurs, but not to as high a level as during the original observation

period. Her present dose varies from 0.1 Gm. twice daily to 0.2 Gm. twice daily, and the blood thiocyanate levels have been between 6.3 mg. and 11.4 mg. per 100 c.c. without any evidence of toxicity.

Fig. 1 demonstrates the variations in blood pressure before and after therapy with thiocyanate.

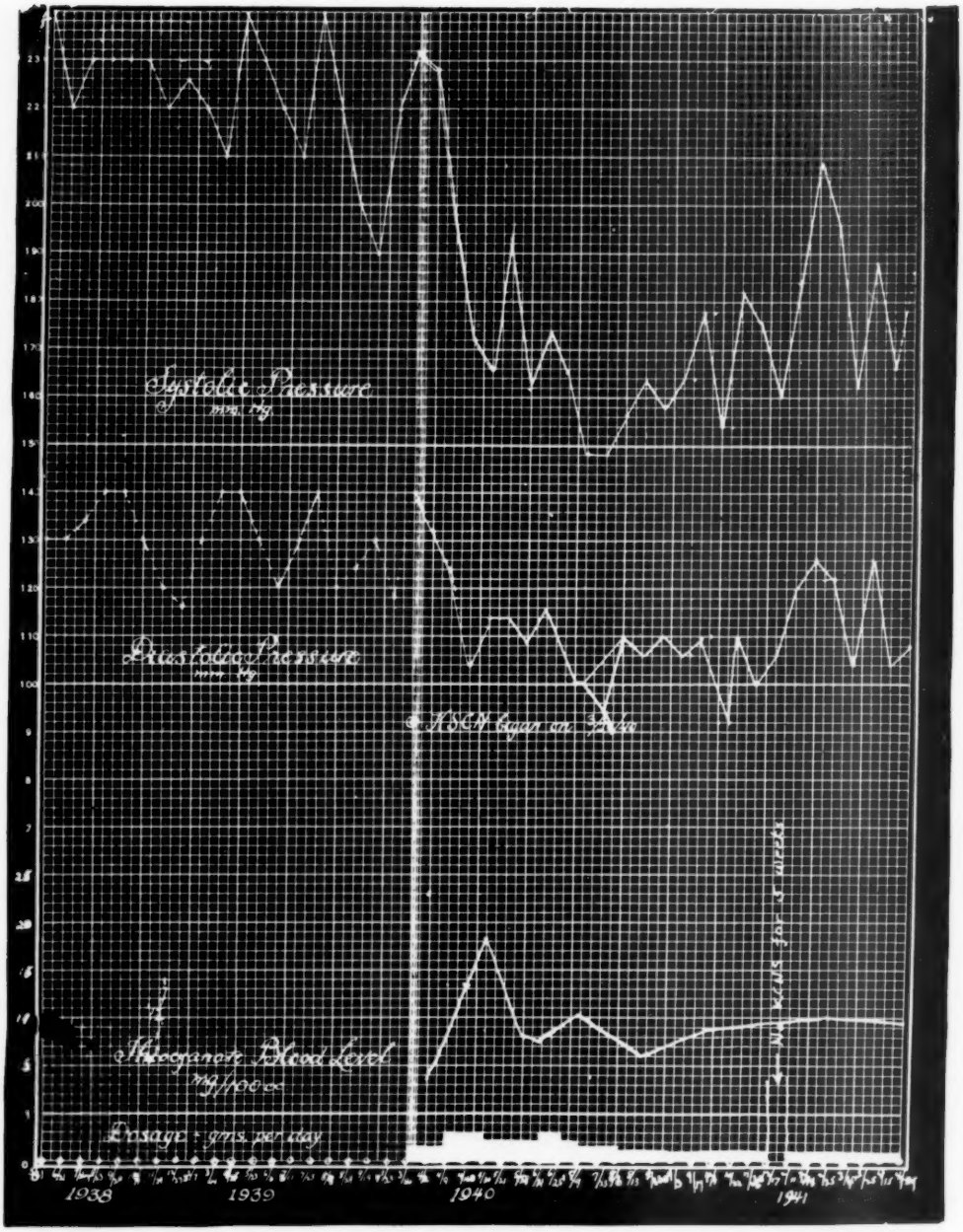

Fig. 1.—Case 1. Systolic and diastolic blood pressure before and after instituting thiocyanate treatment. Daily dose of the drug and blood levels are also shown.

CASE 2.—(Fig. 2.) B. W., a woman, aged 52 years, was admitted to the clinic April 5, 1938, complaining of precordial pain, headaches, dizziness, and some tinnitus. She had just been discharged from the ward, where the diagnoses were: (a) hypertension and arteriosclerosis, (b) enlarged heart, myocardial fibrosis, and coronary sclerosis, (c) regular

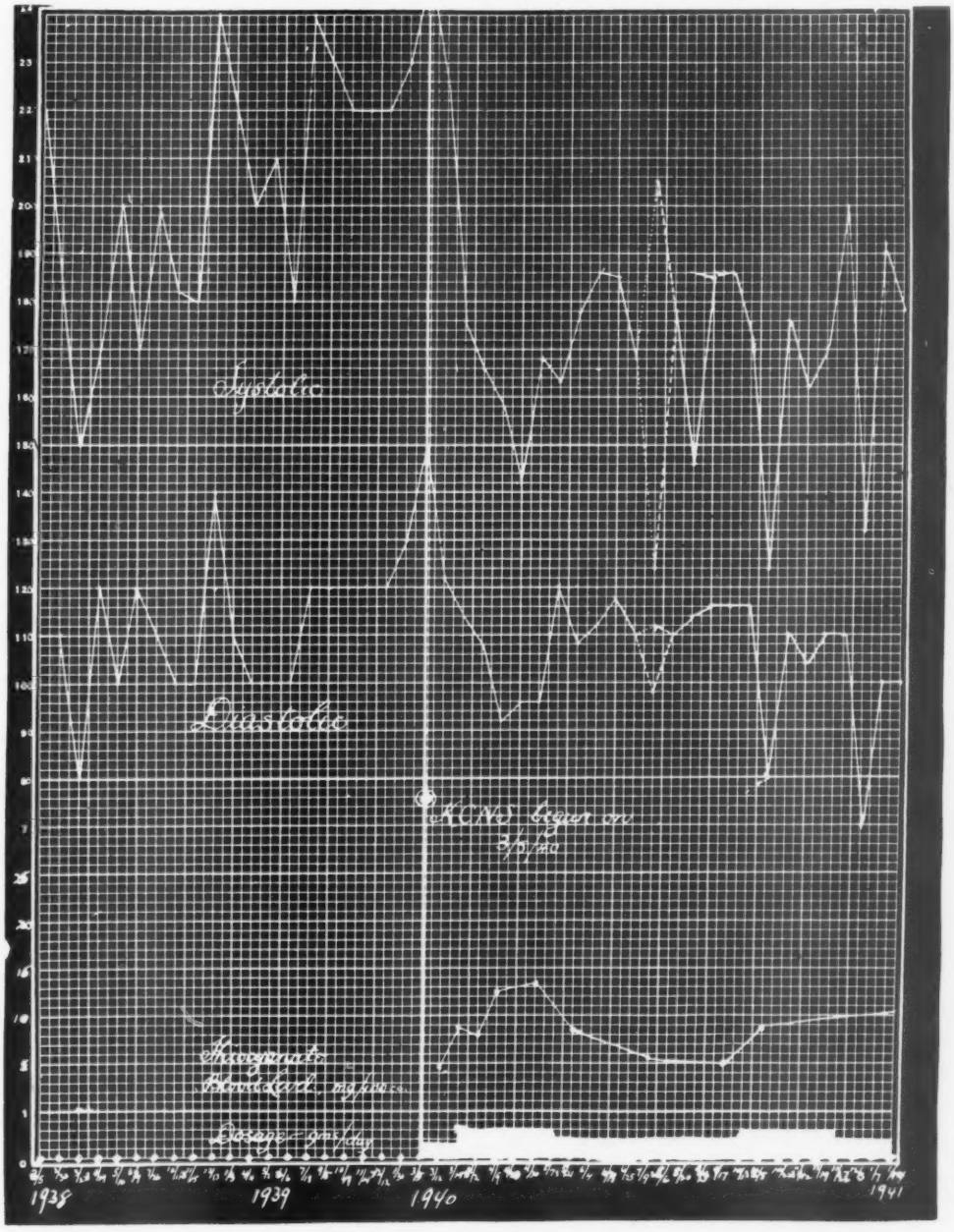

Fig. 2.—Course in Case 2.

sinus rhythm, (d) Class III C, and (e) obesity. The blood pressure on admission was 170/110. The urine concentrated to 1.022 and was entirely normal. The phenolsulfonphthalein excretion was 55 per cent in three hours. The nonprotein nitrogen of the blood was 32 mg. per 100 c.c., and the sugar, 118 mg. per 100 c.c.; the blood Wassermann reaction was negative. The hemoglobin was 75 per cent, the erythrocyte count, 3.3 million per cu. mm., and the leucocyte count, 8,000 per cu. mm., with 60 per cent polymorphonuclear cells. A teleroentgenogram revealed marked enlargement of both cardiac diameters, and the electrocardiogram showed evidence of myocardial damage and deviation of the electrical axis to the left.

She was placed on a 1,000-calorie diet, and was treated with phenobarbital and a mixture of sodium bromide and chloral hydrate. During this time her blood pressure showed marked evidence of fluctuation; the systolic pressure varied from 170 to 240, and the diastolic, from 100 to 140. In addition, the patient, during the period of observation, had numerous, sudden attacks of "fainting," associated with shortness of breath and followed by profuse perspiration and vomiting. There was also evidence of emotional imbalance during the attacks.

Some of these attacks which occurred while she was in the clinic were observed by us, and had no definite pattern. Neurological examination was reported as negative. There was no evidence of gall bladder disease, and the otolaryngologist reported nothing unusual about her vestibular apparatus. It was concluded that these attacks were related to the hypertension, probably a result of encephalopathy.

Potassium thiocyanate therapy was started after an observation period of two years. The initial dose was 0.1 Gm. three times a day. The blood pressure at the time was 250/150. One week later the blood contained 4.4 mg. of thiocyanate per 100 c.c. and the pressure had fallen to 226/122. The same dose of potassium thiocyanate was continued, and the next week the blood pressure was 176/114. The dose was increased to 0.2 Gm. three times daily, and, a week later, the blood pressure had fallen to 166/108 and the blood thiocyanate level was 8.7 mg. per 100 c.c. She has now been maintained on the thiocyanate treatment for almost two years; the thiocyanate level is maintained between 5 mg. and 13.3 mg. per 100 c.c.

This patient still shows marked lability of the blood pressure, with wide fluctuations; the general range, however, is considerably lower. In addition, there has been almost complete cessation of the "fainting" attacks described above; their occurrence has decreased to intervals of several months, whereas previously they had manifested themselves twice and three times weekly.

CASE 3.—(Fig. 3.) B. R., a man, aged 51 years, was first seen in the clinic on Dec. 28, 1937. He was admitted with complaints of generalized, throbbing headaches, poor memory, dizziness, dyspnea, orthopnea, palpitation, precordial pain, nocturia, and fatigue. The blood pressure on admission was 238/130. A teleroentgenogram revealed enlargement of the heart in all diameters, accentuation of the left ventricular curve, and dilatation and tortuosity of the aorta. The electrocardiogram showed deviation of the electrical axis to the left. A pyelogram made at another hospital had been reported as showing some shagginess of the calyces of the right pelvis. Excretion pyelograms made when the patient was admitted to the ward showed that both kidneys were normal in size, shape, and position. The pelves, calyces, and ureters were normal

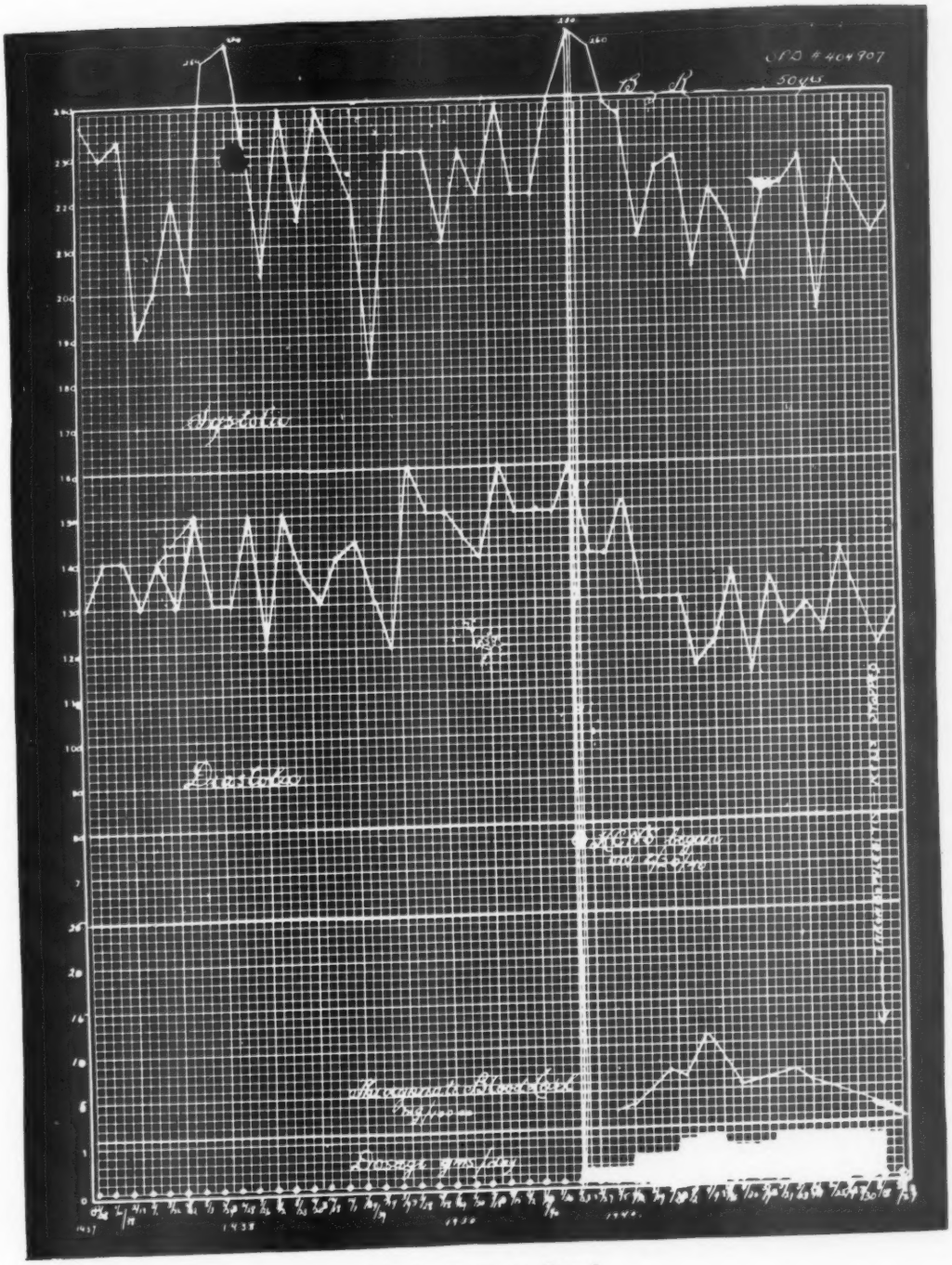

Fig. 3.—Course in Case 3.

on both sides. The electrocardiogram showed sinus rhythm and deviation of the electrical axis to the left. The blood Wassermann reaction was negative, the hemoglobin was 85 per cent, the erythrocyte count was 4.65 million, and the leucocyte count was 6,600 per cu. mm., with 80 per cent polymorphonuclears and 20 per cent lymphocytes. The nonprotein nitrogen of the blood was 31 mg. per 100 c.c., the creatinine, 2, and the uric acid, 3.6. The urine had a specific gravity of 1.018 and contained a trace of albumin. Occasional hyaline and granular casts were seen on microscopic examination, as well as an occasional erythrocyte and 2 to 3 leucocytes per high-power field.

After a period of observation of slightly over three years, during which time the systolic blood pressure was persistently at 190 to 260 and the diastolic at 130 to 150, he was started on potassium thiocyanate therapy. The initial dose was 0.1 Gm. three times a day, and, after a week, a blood level of 4 mg. of thiocyanate per 100 c.c. was reached. The blood pressure, however, remained at 257/140. On the same dose, a week later, the blood pressure was 232/140, and the blood thiocyanate, 4.2 mg. per 100 c.c. Slight dermatitis of the scalp was noted, but it was decided to continue the thiocyanate therapy, and the dose was increased to 0.2 Gm. three times daily. A week later, the thiocyanate level in the blood was 6.9 mg. per 100 c.c., but the blood pressure remained elevated (226/130). The dermatitis had disappeared, and the patient claimed that he was better because the severe headaches had abated, so the dose was increased again to 0.3 Gm. three times daily. There was no increase in the blood thiocyanate; the concentration remained at 6.45 mg. per 100 c.c. The blood pressure was still high, so that he was given 0.4 Gm. three times a day, a total of 1.2 Gm. daily. This time he reached a level of 10.8 mg. thiocyanate per 100 c.c. There was no perceptible lowering of the blood pressure. The dose of 1.2 Gm. daily was continued for two weeks, when he began to have some nausea and loss of appetite, and it was decreased to 0.3 Gm. three times a day, lowering the concentration of thiocyanate in the blood to 5.5 mg. per 100 c.c. The blood pressure had not been lowered at all, but, since the patient stated that he felt better while taking the medicine, it was continued.

Case 4.—(Fig. 4.) M. D., a woman, aged 52 years, was admitted to the clinic Jan. 9, 1940, with headache, giddiness, dyspnea, palpitation, and precordial pain radiating to the left shoulder. The blood pressure was 212/108. Fluoroscopic examination revealed that the heart was normal in size and the chambers normal in contour. The electrocardiogram revealed no abnormalities. The blood nonprotein nitrogen was 36, the creatinine, 1.7, and the uric acid, 3.7 mg. per 100 c.c. The urine specific gravity was 1.015; the urine contained a very faint trace of albumin, and, on microscopic examination, a few clumps of leucocytes were seen. The diagnosis was essential hypertension with no evidence of cardiac involvement.

After a short period of observation, during which the blood pressure was between 204 and 212, systolic, and 108 and 118, diastolic, she was placed on thiocyanate treatment. The usual initial dose of 0.1 Gm. three times daily was given for one week, and she returned to the clinic complaining of nausea, vomiting, loss of appetite, dizziness, and weakness. The blood pressure had fallen to 154/92, and the thiocyanate was found to be 12.5 mg. per 100 c.c. blood. The potassium thiocyanate was discontinued and then started again in a dose of only 0.1 Gm. daily.

The blood pressure has remained low with a dose varying between 0.1 Gm. daily and 0.1 Gm. twice daily; the systolic ranged between 150 and 160, and the diastolic, between 90 and 100. The thiocyanate concentration in the blood with this dose has varied between 5.9 and 7.3 mg. per 100 c.c. Ten months after the thiocyanate had been started she developed a dermatitis, and the drug was stopped, whereupon the blood

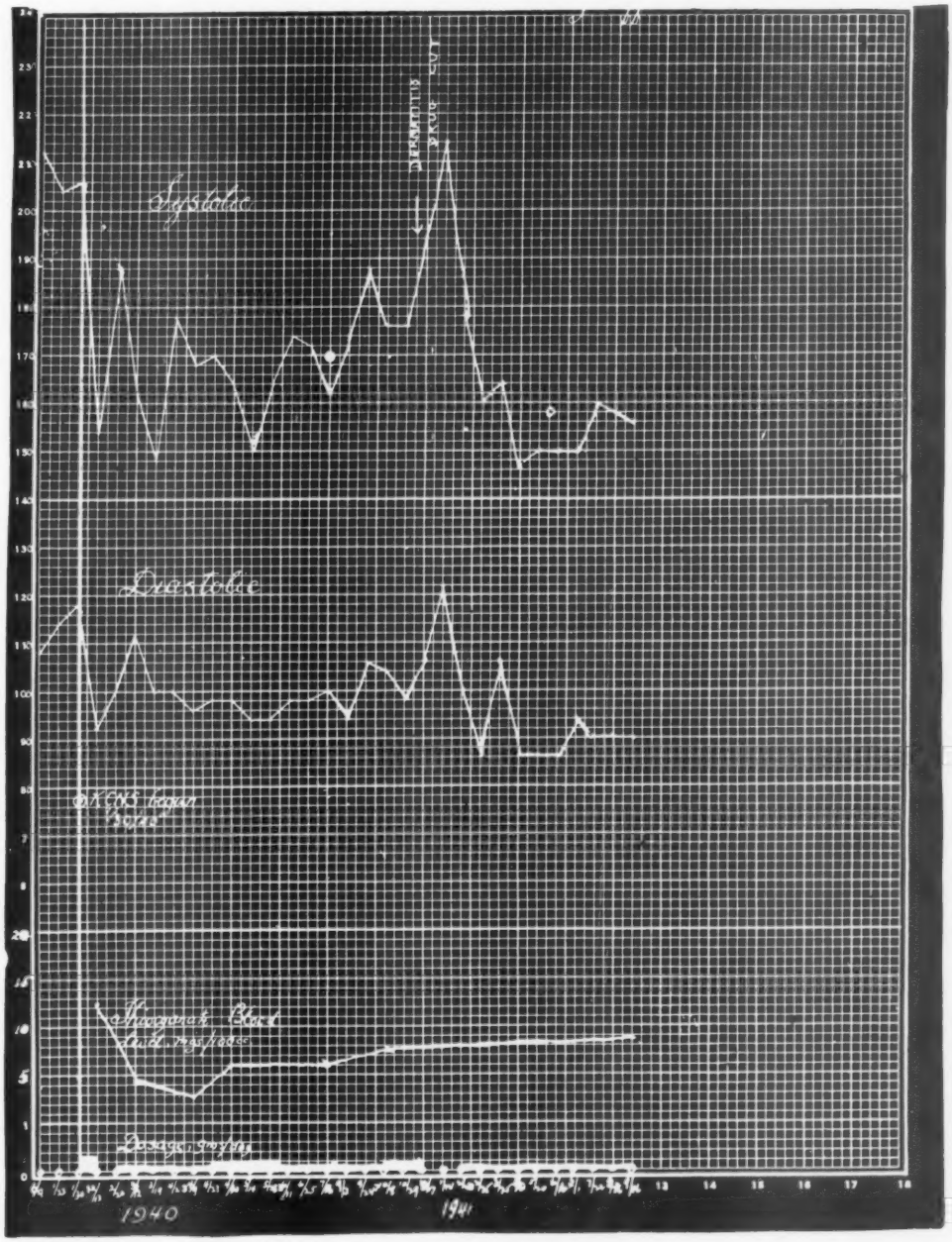

Fig. 4.—Course in Case 4.

pressure rose to 210/120. After the dermatitis cleared up, thiocyanate treatment was started again and the blood pressure fell to the previous low level.

CASE 5.—(Fig. 5.) E. P., a woman, aged 62 years, was admitted to the clinic Oct. 24, 1939, with a history of nocturnal attacks of paroxysmal

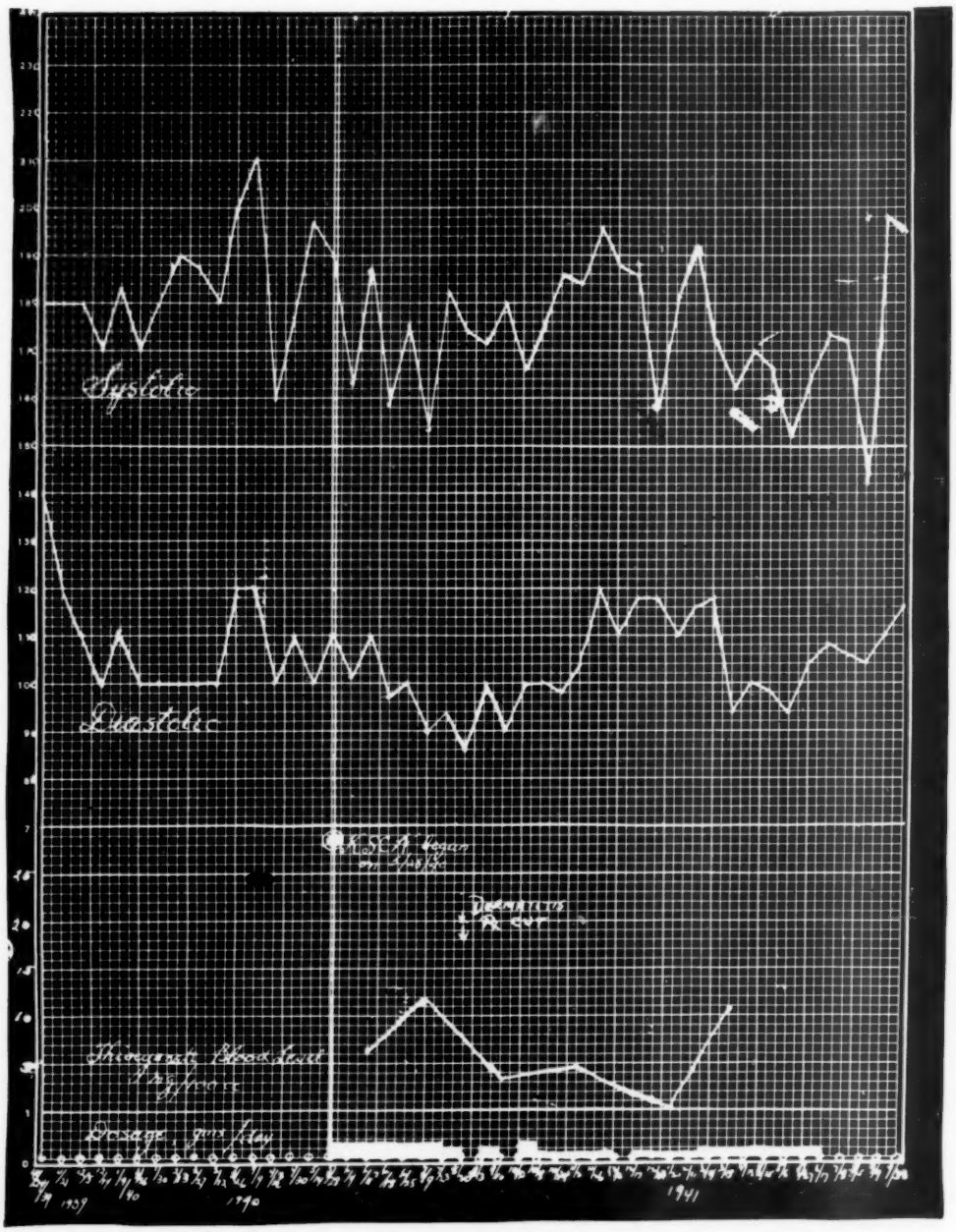

Fig. 5.—Course in Case 5.

dyspnea and substernal oppression of eight months' duration. The heart on roentgenologic examination was enlarged in all diameters; there was accentuation of the left ventricular curve, and the size and shape of the aorta were within normal limits. The blood Wassermann reaction was negative, the nonprotein nitrogen was 45 mg., the sugar, 100 mg., the uric acid, 4.4 mg., and the creatinine, 2.7 mg. per 100 c.c. The urine showed a specific gravity between 1.009 and 1.026, 1 plus albumin, and an occasional granular cast. The electrocardiogram showed regular sinus rhythm and deviation of the electrical axis to the left.

After a control period of seven months, during which the blood pressure ranged between 160 and 210, systolic, and 100 and 140, diastolic, she was started on potassium thiocyanate therapy. In spite of an adequate concentration of thiocyanate in the blood, there was no appreciable lowering of the blood pressure. This can best be appreciated by a study of the chart, in which it is to be noted that, although occasionally there was a lowering of the blood pressure during thiocyanate therapy, similar changes occurred with nonspecific treatment.

COMMENT

These cases have been selected to demonstrate the results obtained in the entire series. Approximately fifty per cent of the patients showed an appreciable lowering of the blood pressure. A few of the patients, as in Case 3, claimed to be subjectively improved while taking the thiocyanate in spite of maintenance of the high level of blood pressure. This is a factor that is very difficult to evaluate, and may be entirely suggestive.

Case 4 illustrates the fact that optimum blood levels can be maintained with comparatively small doses. Moreover, dangerously high blood concentrations may be reached with average doses. The importance of determining the thiocyanate concentration in the blood at fairly frequent intervals during therapy cannot be stressed too strongly.

The marked lability of the blood pressure in all of the cases can be seen from a study of the figures. Although, occasionally, lowering of the blood pressure is observed during treatment, similar instances are seen to have occurred without any specific therapy.

This illustrates the importance of an adequate period of observation of the patient on various methods of treatment before studying the value of any newer form of therapy.

No fatalities and no dangerous toxic effects were observed. This is accounted for by the fact that the patients were seen every week during the first few weeks of thiocyanate therapy, and frequent blood thiocyanate determinations were made. In this way, early toxic symptoms, as well as the appearance of an abnormally high blood level, could be detected and treated before dangerous symptoms supervened.

EXPERIMENTAL WORK

In the course of our studies, it occurred to us that, since the patients were taking the drug in divided doses, blood specimens taken in vary-

ing time relation to the ingestion of the drug might give us different results with respect to the thiocyanate content of the blood. It was decided to study this possibility in the following manner: After the patients had been stabilized with regard to the thiocyanate dosage and blood level, they were asked to come to the laboratory in the morning before taking any of the drug. On arrival at the clinic laboratory, a blood specimen was removed which we termed the "fasting specimen." The patient then took the prescribed dose and specimens were removed at one-, two-, and three-hour intervals. A second dose of the drug was again administered, and blood specimens removed one hour and three hours later. All six specimens were then analyzed for their thiocyanate content. As is evident from a study of the figures in Table I, there was no material change in the blood concentration throughout the twenty-four hours, irrespective of the amount of the drug that the patient was taking.

TABLE I

BLOOD THIOCYANATE LEVELS

CASE NUMBER	FASTING SPECIMEN	ONE HOUR AFTER FIRST DOSE	TWO HOURS AFTER FIRST DOSE	THREE HOURS AFTER FIRST DOSE	ONE HOUR AFTER SECOND DOSE	THREE HOURS AFTER SECOND DOSE
			Mg. per 100 c.c. Blood			
1	15.4	12.9	12.9	12.9	13.8	14.3
2	12.9	12.9	12.9	12.9	12.9	13.8
3	9.7	10.3	10.8	11.4	10.3	10.8
4	18.0	18.0	19.0	19.0	19.3	19.0

Whether or not the blood concentration of thiocyanate varies appreciably throughout the day was checked in still another fashion. This time hospitalized patients were utilized, and blood specimens were taken at 4-hour intervals during the day; that is, at 8:00 A.M., 12 M., 4:00 P.M., and 8:00 P.M., irrespective of the time at which the medicine was taken and regardless of the frequency of administration, whether twice or three times daily.

The results of this study are tabulated in Table II. Here again there was a fairly constant level of the thiocyanate concentration in the blood throughout the day.

TABLE II

BLOOD THIOCYANATE LEVELS AT INTERVALS DURING THE DAY, IRRESPECTIVE OF THE TIME AT WHICH THE MEDICINE WAS TAKEN

CASE NUMBER	8:00 A.M.	12 M.	4:00 P.M.	8:00 P.M.
		Mg. per 100 c.c. Blood		
1	6.9	7.3	4.8	7.3
2	12.1	11.8	12.5	–
3	7.7	9.3	8.2	–
4	8.4	8.7	8.9	8.9
5	5.9	6.3	6.5	6.3
6	4.7	5.3	5.0	5.6

The constancy of the blood level made it appear that absorption and excretion occurred at equal rates. We therefore decided to study this problem further by comparing the daily twenty-four-hour urinary output of the drug with the daily intake.

URINARY EXCRETION OF THIOCYANATE COMPARED WITH DAILY INTAKE

For the purpose of this study, six patients were hospitalized so that they could be observed closely and all of their urine collected. The dose of thiocyanate in Cases 1, 2, 3, and 4 had previously been ascertained, so that an optimum blood concentration had been reached. In Cases 5 and 6, as can be seen from the rising blood concentration, the patients had just started on the drug. In all patients, a daily blood concentration was determined, and the entire urinary output in each twenty-four hours was collected separately. By filtering the urine through animal charcoal, a clear solution was obtained, and the concentration of thiocyanate was easily determined. The daily output could then be calculated.

TABLE III

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
8/ 7/41	0.4	239	7.4			
8/ 8/41	0.4	239	7.4	1,100	11.8	129.8
8/ 9/41	0.4	239	8.4	1,900	11.8	224.2
8/10/41	0.4	239		1,500	4.3	64.5
8/11/41	0.4	239		1,400	6.5	91.0
8/12/41	0.4	239	8.0	1,900	4.9	93.1
8/13/41	0.4	239	7.9	1,400	4.5	63.0
8/14/41	0.4	239	8.4	1,000	3.7	37.0

*The subject of this study was B. W., a woman, aged 51 years (Case 1).

In Case 1 (Table III), on a constant daily dose of 0.4 Gm. of potassium thiocyanate, or 239 mg. calculated as thiocyanate ion, a fairly constant blood concentration was maintained; the variation was from 7.4 to 8.4. However, marked variation was found in the urinary output, namely, from as low as 37 mg. to as high as 224.2 mg.

In Case 2 (Table IV), a fairly constant blood level was also maintained; the range was from 6.3 to 8.7 mg. per 100 c.c. Here, again, a marked variation in urinary output was found; the figures ranged from 54.6 mg. to 287.5 mg.

In Case 3 (Table V), such marked variation in excretion was not found, but the amount excreted was only a small part of the amount ingested.

In Case 4 (Table VI) there was also marked variation in the urinary thiocyanate output.

TABLE IV

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
7/28/41	0.2	358	8.0			
7/29/41	0.6	358	8.1	515	15.4	79.3
7/30/41	0.6	358	8.7			
7/31/41	0.6	358	8.0	1,150	18.2	209.3
8/ 1/41	0.6	358	7.1	700	30.8	215.6
8/ 2/41	0.6	358	6.3	1,150	25.0	287.5
8/ 3/41	0.6	358		375	16.7	62.6
8/ 4/41	0.6	358	6.5	300	18.2	54.6
8/ 5/41	0.6	358	6.7	700	16.7	116.9
8/ 6/41	0.6	358	7.7	850	20.0	170.0

*The subject of this study was B. G., a woman, aged 65 years (Case 2).

TABLE V

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
7/30/41	0.2	120	7.7			
7/31/41	0.2	120	6.9			
8/ 1/41	0.2	120	6.3	500	13.3	66.5
8/ 2/41	0.2	120	6.1	350	20.0	70.0
8/ 3/41	0.2	120		478	12.2	58.3
8/ 4/41	0.2	120	5.1	325	13.3	43.2
8/ 5/41	0.2	120	5.1	600	11.8	70.8
8/ 6/41	0.2	120	4.9	400	10.5	42.0
8/ 7/41	0.2	120	5.4	425	9.3	39.5
8/ 8/41	0.2	120	4.7	400	7.4	29.6

*The subject of this study was M. K., a woman, aged 47 years (Case 3).

TABLE VI

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
7/31/41	0.3	179	6.9			
8/ 1/41	0.3	179	6.1	1,650	8.2	135.3
8/ 2/41	0.3	179	6.1	1,050	5.0	52.5
8/ 3/41	0.3	179		1,050	8.7	91.3
8/ 4/41	0.3	179	6.3	950	3.5	33.2
8/ 5/41	0.3	179	5.9	1,400	3.5	49.0
8/ 6/41	0.3	179	5.7	1,600	4.6	73.6
8/ 7/41	0.3	179	6.5	1,600	5.4	86.4
8/ 8/41	0.3	179	5.9	1,600	5.3	84.8

*The subject of this study was M. J., a woman, aged 63 years (Case 4).

TABLE VII

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
8/ 7/41	0.3	179	1.7	1,725	2.1	36.2
8/ 8/41	0.3	179	2.4	1,850	3.0	55.5
8/ 9/41	0.3	179	2.7	2,400	2.1	50.4
8/10/41	0.3	179		1,800	4.3	77.4
8/11/41	0.3	179				
8/12/41	0.3	179	4.3	950	9.1	86.4
8/13/41	0.3	179	4.5	2,000	3.7	74.0
8/14/41	0.3	179	5.0	2,000	3.6	72.0
8/15/41	0.3	179	4.7		5.3	
8/16/41	0.3	179	5.3	1,870	4.7	87.9
8/17/41	0.3	179		2,000	3.9	78.0
8/18/41	0.3	179	6.9	2,000	5.1	102.0

*The subject of this study was B. R., a man, aged 51 years (Case 5).

TABLE VIII

COMPOSITE STUDY* OF DAILY INTAKE OF THIOCYANATE, THE BLOOD CONCENTRATION, AND THE URINARY EXCRETION

DATE	DAILY DOSE		BLOOD CONCENTRATION	URINE		
	AS KCNS. (GM.)	AS CNS. (MG.)		VOLUME (C.C.)	MG. CNS. PER 100 C.C.	TWENTY-FOUR-HOUR OUTPUT (MG.)
7/28/41	0.3	179	1.8			
7/29/41	0.3	179		550	3.3	18.1
7/30/41	0.3	179	3.6	480	25.0	120.0
7/31/41	0.3	179	3.3			
8/ 1/41	0.3	179	4.0	960	5.5	52.8
8/ 2/41	0.3	179	4.3	1,020	5.6	57.1
8/ 3/41	0.3	179		900	5.6	50.4
8/ 4/41	0.3	179	5.4	1,080	6.9	74.5
8/ 5/41	0.3	179	5.3	640	9.3	59.5
8/ 6/41	0.6	358	5.6	420	6.7	28.1
8/ 7/41	0.6	358	6.1	600	8.2	49.2
8/ 8/41	0.6	358	7.1	700	6.3	44.1
8/ 9/41	0.6	358	11.1	600	6.5	39.0
8/10/41	0.6	358		600	5.9	35.4
8/11/41	0.6	358	8.6	600	4.3	25.8
8/12/41	0.6	358	11.1	650	8.6	55.9
8/13/41	0.6	358	12.1	175	15.4	26.9

*The subject of this study was I. L., a man, aged 58 years (Case 6).

In Cases 5 and 6 (Tables VII and VIII), the patients had been started on the drug. As can be seen, the blood concentration shows a steady rise to the optimum level. In Case 6, an increase in the daily dose was required. Here again no correlation between urinary excretion and the dosage was found.

This marked variation in the urinary output is rather difficult to explain in view of a fairly constant maintenance of the blood level. We must conclude that other excretory routes are probably utilized. Goldring and Chasis,[30] in comparing the urinary output of thiocyanate with

the daily intake, without following the blood levels, also concluded: "there appears to be a distinct lack of relationship between the average amount excreted per day and the average administered per day."

DISCUSSION

The mechanism of the action of the thiocyanates in the reduction of blood pressure is not clearly understood. Healy[25] observed a marked lowering of blood pressure in his cases, and, in some experiments on rabbits, obtained a strongly positive reaction for thiocyanate in the adrenal cortex. This led him to conclude that the effect is similar to a hypoadrenia. Lindberg, Wald, and Barker[16] gave toxic doses of thiocyanate to dogs, and obtained blood levels of 20 to 60 mg. per 100 c.c. They found a marked lowering in blood cholesterol, a fall in blood proteins, a definite fall in hemoglobin, hematocrit reading, and erythrocyte count, an aplastic condition of the bone marrow, and fatty degeneration of the liver. Routine examination of the myocardium, kidneys, spleen, pancreas, lungs, and thyroid showed no gross or microscopic alterations. Anatomic changes in the adrenals were lacking. Analysis showed no greater amount of thiocyanate in this organ than in the others. Blood studies in reference to sugar metabolism, sodium, potassium, and chlorides failed to disclose any evidence of change in adrenal function. They concluded: "We are unable to support the impression of Healy that hypoadrenia may be the principal feature of the toxicology of the thiocyanates."

The recent work of Caviness and his associates[24] is of interest. They report that thiocyanates are naturally present in the body in a much higher concentration than any other known depressor substances. They found a natural concentration of thiocyanate in 241 persons to whom the drug had never been administered of 0.31 to 2.55 mg. per 100 c.c. of blood, and that the blood pressure varied in inverse ratio to the blood thiocyanate concentration. They suggest that the thiocyanates help to counterbalance the effects of the pressor substances in the body, and conclude that the use of thiocyanates in hypertension need no longer be regarded as empirical.

Despite the accumulation of reports of thousands of patients treated with thiocyanate without fatality, death occasionally occurs, as in the case reported recently by Russell and Stahl.[27] Extreme caution should be used in selecting patients for treatment. Their patients would seem to have been a poor choice because of the greatly diminished kidney function, as evidenced by a urea clearance of 27 per cent of normal, a nonprotein nitrogen of 43 mg. per 100 c.c. of blood, and 3 plus albuminuria. Furthermore, poor judgment was shown in continuing the administration of thiocyanate to their patient when a level of 15.2 mg. had been reached. This is particularly important in view of the fact that the dose had not been changed, and that one week previously his blood level had been 4.5 mg. Such a rapid rise in blood concentration

is certain evidence of faulty excretion and cumulative action, and indicates immediate cessation of the drug. One can only conclude, as do Mandelbaum[28] and Ayman,[29] that this was an unfortunate choice of a patient, rather than a condemnation of the drug.

SUMMARY AND CONCLUSIONS

1. Thirty-nine ambulatory patients with hypertension were treated with thiocyanate. Approximately fifty per cent showed an appreciable lowering of the blood pressure, and a few others reported subjective improvement without any lowering of the blood pressure.

2. Ambulatory and hospital patients who were receiving potassium thiocyanate for hypertension were studied to ascertain whether the blood concentration of the drug varied during the day, or was affected by the relation of the time of ingestion to the time of taking the blood.

3. The blood level of thiocyanate remains fairly constant throughout the day.

4. Further studies were made to ascertain whether any relation existed between the daily intake or the blood concentration, on one hand, and the urinary output, on the other.

5. There is no correlation between the urinary output of thiocyanate and the daily intake or the blood concentration.

6. The exact mode of action of thiocyanate in hypertension is not clearly defined, and should be the subject of further investigation.

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POSTERIOR BASAL CARDIAC INFARCTION AFFECTING A YOUNG MAN, FOLLOWED A YEAR LATER BY ANTERIOR APICAL INFARCTION

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THE incidence of coronary thrombosis among young adults has become of increasing importance in recent years. Accordingly, we are reporting a case in which two attacks of coronary thrombosis took place just a year apart, the initial one when the patient was 39 years of age, and the second when he was 40 years. In addition, we have sought to collect the total number of cases of coronary occlusion among persons less than the age of 41 years which have been reported to date. Furthermore, we are publishing the electrocardiograms in our case because seldom is so distinct a demonstration available of the role which electrocardiography plays in determining the presence of myocardial infarction, its location, and the existence of the myocardial damage which remains after the acute process has progressed to healing.

In 1939, Master, Dack, and Jaffe,[1] reviewed 500 cases of myocardial infarction due to coronary thrombosis, including thirty-nine cases (7.8 per cent) in which the patients were less than the age of 40 years. Also, they cited literature including 221 instances of this condition among relatively young patients. Of these 221 patients, 106 were less than 40 years of age and 115 were less than 41 years. Inasmuch as none of their references bore a date later than 1938, our survey of the literature concerning coronary occlusion among persons less than 41 years of age has covered the years 1939 to 1943, inclusive. The series of Buchanan, Indelicato, and Primavera,[2] reported in 1939, included seven patients less than the age of 41 years. In the same year, Goodson and Willius[3] cited thirty cases of coronary thrombosis among patients less than the age of 40 years. In a study published in 1942, based on 100 patients, Smith, Sauls, and Ballew[4] found five who were less than 40 years of age. Bancker,[5] also in 1942, reported a series including five patients less than 40 (or 41, possibly) years old. Six isolated reports of cases[6-11] have appeared since 1939; all six of the patients were in the third decade of life. In reviewing cases encountered at the Mayo Clinic since 1939, we found that five cases of undoubted coronary occlusion, including the case we are reporting, occurred among patients less than 40 years of age; two of these patients were 38 and three were 39 years of age. Moreover, two reports of cases[12, 13] in foreign journals were listed in the *Quarterly Cumulative Index Medicus*, but copies of

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the journals were not available to us. Exclusive of the latter, as far as our search disclosed, 318 cases of coronary occlusion among persons less than the age of 41 years have been reported to date. (Since this paper was written, French and Dock[14] have reported data on eighty cases of fatal coronary arteriosclerosis in young persons between the ages of 20 and 36 years.)

REPORT OF CASE

On March 9, 1942, a man, aged 39 years, who gave no history of angina pectoris or hypertension, was admitted to the hospital complaining of severe, constricting substernal pain which had been present intermittently all of the previous night to such degree that he had been unable to sleep.

On examination, the patient was in obvious distress and had moderate pallor, but he exhibited no signs of shock. Results of examination of the lungs were negative. Examination of the heart revealed sounds of somewhat diminished intensity, normal rhythm, and a rate of 90 beats per minute. The blood pressure was 124/90. There were no other physical signs of significance. The leukocytes numbered 14,500 per cubic millimeter of blood. The erythrocyte sedimentation rate was 30 mm. at the end of one hour (Westergren). An electrocardiogram on the day of admission (Fig. 1, *a*) was characteristic of infarction of the posterior basal portion of the left ventricle.

During the next five days the patient's temperature was elevated, and sometimes reached 101° F. The erythrocyte sedimentation rate rose to 47 mm. in one hour on the third day, and 110 mm. in one hour on the fourth day. The electrocardiograms continued to show a classical Q_3T_3 pattern. By March 11, partial (2:1) heart block had developed, with a P-R interval of 0.26 second (Fig. 1, *b*, Lead III). This was still present the following day, although the P-R interval had shortened to 0.24 second. The block had disappeared by March 14. It was believed that the patient had suffered a massive myocardial infarction and that the prognosis was grave. However, after the first week, his recovery was rapid. The erythrocyte sedimentation rate had dropped to 55 mm. in one hour by March 24. The abnormality of the S-T segment progressively subsided (Fig. 1, *c*). The electrocardiogram on March 27 (Fig. 1, *d*) showed a typical late Q_3T_3 pattern. The patient was dismissed from the hospital on March 30.

The man was seen at intervals during the ensuing year, and his progress seemed satisfactory. In electrocardiograms taken on May 15, and July 16, 1942, and on Feb. 24, 1943 (Fig. 1, *e*), there was no deviation from the late Q_3T_3 pattern.

On March 13, 1943, the patient, now aged 40 years, was awakened from sleep at 5:00 A.M. by pain, without extension, in the left shoulder. Shortly, however, he began to have a bandlike constricting pain in the substernal region and the epigastrium; the pain became progressively severe. He was seen at 8:00 A.M. and was given ¼ grain (0.016 Gm.) of morphine, followed by 1/100 grain (0.00065 Gm.) of glyceryl trinitrate, neither of which relieved the pain. On admission to the hospital, the heart tones were of good quality, the cardiac rate was 90 beats per minute, and the rhythm was regular. The blood pressure was 120/84. The lungs were negative. The leukocytes numbered 19,000 per cubic millimeter of blood, and the erythrocyte sedimentation rate was 19 mm. in one hour. The electrocardiogram (Fig. 1, *f*), compared to the last

previous tracing (Fig. 1, *e*), revealed marked decrease in the amplitude of the T waves in Leads I, IVR, and CR_2, and diminution in the depth of the inverted T waves in Leads II and III.

The patient was still in considerable distress at noon, seven hours after the onset, and repeated doses of morphine were required to control the pain. Vomiting occurred several times in the first few hours. With morphine and oxygen, the man was kept reasonably comfortable and the pain had subsided by midafternoon. It was believed that he had suffered a secondary coronary occlusion, and that this time the infarction was involving the anterior apical portion of the left ventricle, as indicated by the electrocardiographic changes.

On March 14, 1943, the heart tones were of poor quality, there was gallop rhythm, a loud pericardial friction rub was audible, and the apex had moved out beyond the nipple line. These signs suggested marked dilatation of the left ventricle. The blood pressure was 110/80. There were moist râles at the bases of the lungs. Treatment with digitalis

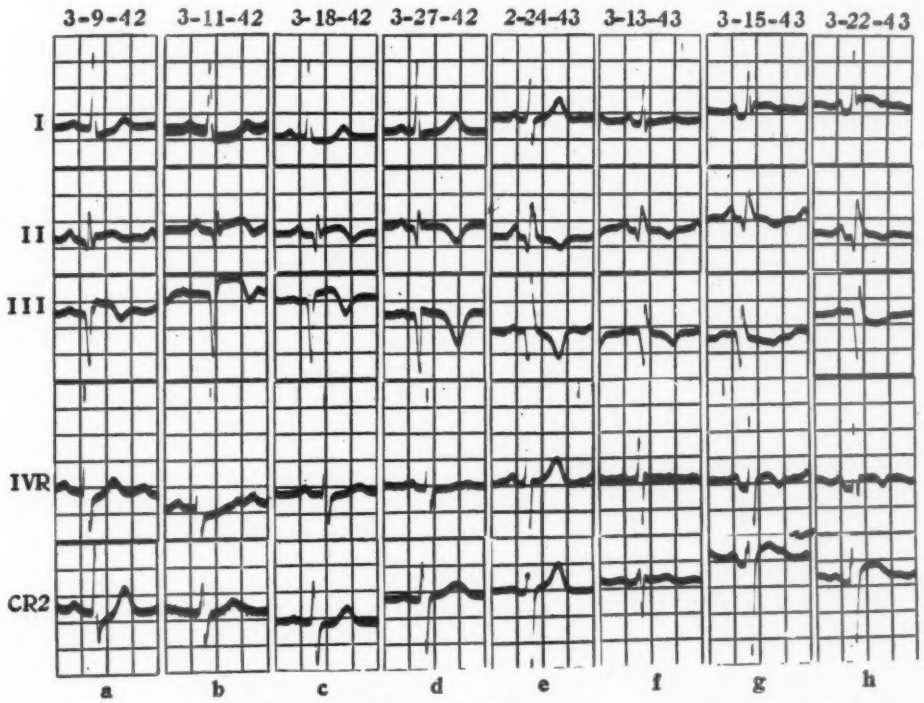

Fig. 1.—Electrocardiographic patterns indicative of posterior basal infarction of the left ventricle, followed a year later by anterior apical infarction:

a, Lengthened Q_3; elevated S-T_2 and S-T_3, depressed S-T_1; inverted T_3 and diphasic T_2; depressed S-T segment in Leads IVR and CR_2.

b, Delayed A-V conduction (P-R, 0.26 second in Lead III); T_2 now frankly inverted.

c, S-T segments returning toward isoelectric line.

d, Further progress of S-T segments toward isoelectric level. Note increased amplitude of inverted T_2 and T_3, and reciprocal, increased positivity of T_1.

e, Seventeen days before second occlusion. Typical late Q_3T_3 pattern

f, Tracing on day of second occlusion. Marked decrease in amplitude of T in Leads I, IVR, and CR_2; also, decreased negativity of T_2 and T_3.

g, Elevated S-T segments in Leads I and CR_2, depressed S-T_3; T inverted in Leads I and IVR; decreasing negativity of T_2 and T_3.

h, S-T segment deviations more marked in all leads. Pattern is now quite typically that of acute anterior apical infarction superimposed on the previous posterior basal infarction.

was instituted. On the next day, the erythrocyte sedimentation rate had risen to 80 mm. in one hour.

On March 22, severe anterior thoracic pain recurred and extended into both arms. This symptom was accompanied by pallor and sweating, and the pulse became feeble and rapid. The blood pressure was 86/72. It was believed that the infarct had extended and that a fatal issue was inevitable. However, on the following day the patient appeared definitely better; the heart tones were of better quality and the rate was slower. This improvement continued, although the erythrocyte sedimentation rate rose to 106 mm. in one hour on March 26.

After the attack of March 13, 1943, the electrocardiograms had continued to show a progression of changes indicating that the pattern of infarction of the anterior apical portion of the left ventricle was being superimposed on that of the previous posterior basal infarction. In the tracing on March 15 (Fig. 1, *g*), there had been an elevated S-T segment and an inverted T wave in Lead I, depression of the S-T segment in Lead III, and flattening of the previously inverted T waves in Leads II and III, and the outstanding changes in the chest leads consisted of an inverted T wave in IVR and elevation of the S-T segment in CR_2. All of these changes had been more marked in the tracing taken on March 22 (Fig. 1, *h*).

On April 11, the patient complained of vague pains in the anterior part of the thorax which were worse with inspiration and limited the respiratory excursions. He had a chill and his temperature rose to 101° F. That evening there were physical signs of consolidation over the lower part of the left lung, posteriorly. The leukocyte count was 20,600 per cubic millimeter of blood. A roentgenogram of the thorax revealed opacity over the lower half of the left pulmonary field, with evidence of some fluid. These abnormalities were interpreted as being due, in all probability, to consolidation subsequent to pulmonary embolism and infarction. The patient tolerated this complication unusually well; there was no febrile reaction after the first day, and specific treatment was not necessary. The physical signs gradually resolved.

The remainder of the patient's stay in the hospital was uneventful, as gradual improvement took place. On April 19, the erythrocyte sedimentation rate had dropped to 44 mm. in one hour. The heart remained enlarged, with the apex beat visible in the anterior axillary line. A loud, blowing, systolic murmur developed and persisted at the apex. Electrocardiograms taken later than those shown in Fig. 1 revealed a combination of the late Q_3T_3 and late T_1 patterns; the S-T segments in all leads had returned to the isoelectric level. The patient was dismissed from the hospital on May 7. At the time this report was written he was able to be up and about a small part of the time, but he had practically no cardiac reserve and he was close to congestive failure.

COMMENT

Hypertension and angina pectoris may be of less frequent occurrence in the histories of relatively young patients with coronary thrombosis than in the histories of older patients with this condition. Clawson[15] found that, among forty-eight patients less than 40 years of age who had coronary sclerosis severe enough to cause death, 19 per cent had accompanying hypertension, whereas, among those 40 years of age or

more, 46 per cent had hypertension. In White's[16] series there was no hypertension among patients (all males) less than 40 years of age, contrasting with a 25 per cent incidence of hypertension among patients of all ages. White found angina pectoris preceding coronary thrombosis among only 14 per cent of patients less than the age of 40 years, whereas, of 125 patients of all ages who had angina pectoris associated with coronary thrombosis, 76 per cent had angina before the thrombosis.

Smith, Sauls, and Ballew observed cardiac enlargement in 59 per cent of their cases of coronary thrombosis. Master, Dack, and Jaffe found this in 59.8 per cent of patients of all ages, but noted further that the incidence of cardiac enlargement was only 35.9 per cent among patients less than 40 years of age, and that, regardless of age, its incidence closely paralleled that of heart failure, which is the principal cause of death (considering all age groups) after coronary occlusion.

Smith, Sauls, and Ballew noted embolic phenomena in 10 per cent of their cases. Here again, Master, Dack, and Jaffe made a critical analysis which is illuminating. They observed that, among patients less than 50 years of age who suffer coronary thrombosis, the most frequent cause of death was arterial embolism, either peripheral or pulmonary; the death of 53 per cent of the patients in this age group was due to this cause, whereas the mortality due to this complication among those aged 50 years or more was only 12 per cent, and the cause of death was much more likely to be heart failure.

SUMMARY

According to our search of the literature, 318 cases of coronary thrombosis among patients less than 41 years of age have been reported. If we were able to validate two additional cases reported in foreign journals, the total would be brought to 320.

Our patient, whose case is here reported, suffered two attacks of coronary thrombosis. The first attack occurred at the age of 39 years, and resulted in infarction of the posterior basal portion of the left ventricle; the second occurred at the age of 40 years, and caused infarction of the anterior apical portion. The electrocardiograms depict the accuracy with which this diagnostic aid may reflect the presence and the location of myocardial infarction.

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ASSOCIATION OF HYPERTENSION AND MITRAL STENOSIS

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CERTAIN special relationships have been suggested regarding the development of hypertension in persons who have mitral stenosis. The first of these is that elevated blood pressure tends to ameliorate the clinical course of the valvular disability. Levine,[1] in considering this association, summarizes his views as follows: "Although it may add certain symptoms as a result of the hypertension itself, when these are not severe, there is reason to believe that it delays the progress of mitral stenosis. It is curious that so many patients with mitral stenosis over fifty years of age have hypertension in addition. I believe that hypertension prolongs their lives and enables them to reach the latter decades."

Levine suggests two factors as being possibly responsible for the beneficial effect which he notes. One is that the hypertension tends to induce and maintain enlargement and dilatation of the left ventricle, and that this dilatation opposes and delays the contraction of the mitral ring. The second factor which he considers is that the superimposition of hypertension upon mitral stenosis equalizes the burdens upon the two sides of the heart, and thus delays the development of an imbalance between them.

In addition to this salutory influence, it has been suggested that persons with mitral stenosis develop hypertension with greater frequency than do those of the general population. Levine feels that such an increased incidence occurs in the age range beyond 45 years, but that there is a tendency toward decreased blood pressure in the younger group of persons with mitral disease. He considers that a possible explanation is that the chronic rheumatic infection responsible for the valvular lesion may also insidiously produce widespread vascular damage which later results in hypertension. However, he thinks it more likely that certain persons have what he calls "vascular vulnerability," which renders them more susceptible to both rheumatic fever and the later development of hypertension.

Although the first of these two relationships is of greater clinical interest and largely conditions the interest in the latter, it will receive only subsidiary consideration here because of the limitations of the material available. The primary problem with which the present study is concerned is the relative frequency of the development of hypertension in persons with mitral stenosis.

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The literature regarding the question is not voluminous. Boas and Fineberg,[2] in reviewing it in 1926, found that Bamberger, in 1857, had noted a frequent association of valvular heart disease and granular kidneys. Goodhart, in 1880, described the frequent occurence of granular kidneys in patients with mitral thickening and stenosis. Pitt, in 1888, surveying a large necropsy material, reported that one-third of females and one-fifth of males with mitral stenosis also had granular kidneys. Both Gibson and Coombs had noted the common occurrence of high arterial pressure in patients with mitral stenosis.

In conjunction with this review of the literature, Boas and Fineberg[2] reported a study of the blood pressure in 135 cases of mitral stenosis. Using a blood pressure in excess of 150, systolic, and 90, diastolic, as a criterion of hypertension, they found an incidence of 29 per cent (thirty-nine cases) in their total group whose age range was from 1 year to over 60 years of age. Sixty-seven of these were over 40 years of age, and, in this age range, the incidence of hypertension was 55 per cent. They compared these frequencies with the 11 per cent incidence in a group of 3,778 hospital patients who were unselected for age and sex.

Levine and Fulton[3] studied a series of 762 cases of mitral stenosis in which the ages ranged from 5 to over 70 years. One hundred fifty-nine of these patients were over 45 years, and, of them, ninety-two, or 58 per cent, had hypertension by the same criterion as that employed by Boas and Fineberg. Their conclusion was that "with advancing years patients with mitral stenosis have vascular hypertension with much greater frequency than the average population."

Brumm and Smith,[4] however, in reviewing 1,925 patients of all ages at the Mayo Clinic, found that only forty-four, or 2.3 per cent, had hypertension associated with mitral stenosis. Although their criterion of hypertension was not mentioned, it seems reasonable to assume from the fact that the lowest blood pressure in their hypertensive group was 152, systolic, and 90, diastolic, that it was approximately the same as that employed in the other two studies. They further concluded that the hypertension in these patients exerted no beneficial effect.

The material for the present study consists of cases of proved mitral stenosis, taken from the autopsy records of the pathology department of the University of Minnesota Medical School. Because of the possible mechanical effects of aortic valvular lesions upon blood pressure, all cases in which there was accompanying aortic valve involvement were excluded. However, in some instances there was an associated mitral regurgitation. All were cases of persons who died in Twin City hospitals during the period between 1929 and 1940. The blood pressures were taken from the hospital charts, either directly or as recorded on the autopsy reports; in those instances in which more than one blood pressure reading was recorded, the highest value was taken. Cases in which the patient was in coma or in which the blood pressure was taken within

twelve hours of death were excluded. The final group consisted of 144 cases with an age range of 20 to 87 years; forty-three (30 per cent) were males, and 101 (70 per cent) were females.

As a control group, a stratified sample of 288 cases, exactly matching the mitral group with respect to age and sex, was taken at random from the records of the out-patient clinic of the University Hospital. The blood pressures of these patients were those recorded at their initial physical examinations in the clinic.

Following the precedent of the studies noted above, a blood pressure in excess of 150, systolic, and 90, diastolic, was taken as the arbitrary criterion of hypertension. Because of the emphasis upon the factor of age by Levine and Fulton, their procedure of dividing the total group into two subgroups, with the age of 45 years as the point of division, was also employed here.

These conditions afford two bases of comparison between the mitral and control groups and between their respective subgroupings. They are first compared with regard to the frequency of the arbitrarily defined hypertension, and, second, on the basis of the average values for systolic and diastolic pressures.

The results of the first comparison for the total groups, for the subgroup over the age of 45, and for the subgroup under the age of 45 years are summarized in Table I. The differences observed are, in all cases, slight; the greatest critical ratio was 1.35, and therefore they fail to satisfy any of the criteria of statistical significance. In particular, the frequencies in which we are the most interested, namely, those for the age range over 45 years, are nearly identical for the mitral and control groups. As noted, the values are 30 and 31 per cent, respectively.

TABLE I

INCIDENCE OF HYPERTENSION IN MITRAL AND CONTROL GROUPS

	TOTAL		OVER AGE 45		UNDER AGE 45	
	MITRAL	CONTROL	MITRAL	CONTROL	MITRAL	CONTROL
Number of Cases	144	288	91	182	53	106
Number with Hypertension	34	62	27	56	7	6
Percentage with Hypertension	23.6	21.6	30	31	13	6
Standard deviation	3.6	2.4	4.9	3.4	4.6	2.3
	Difference 2		Difference 1		Difference 7	
	S. D. difference 4.33		S. D. difference 5.96		S. D. difference 5.13	
	Critical ratio .46		Critical ratio .17		Critical ratio 1.35	

This observed difference of 1 per cent is actually in the reverse direction from that expected and has a standard deviation of 5.96, which means that the chances that such a difference would occur by the accident of random sampling are about eighty-five in a hundred.

TABLE II

AVERAGE SYSTOLIC BLOOD PRESSURES FOR CONTROL AND MITRAL GROUPS

	TOTAL	OVER AGE 45	UNDER AGE 45
Control	139.86 ± 1.68	148.2 ± 2.34	125.5 ± 1.79
Mitral	138.9 ± 2.28	144.4 ± 3.02	129.6 ± 2.88
Difference	.96	3.8	4.1
S. D. difference	2.78	3.81	3.39
Critical, ratio	.34	1	1.2

TABLE III

AVERAGE DIASTOLIC PRESSURES FOR CONTROL AND MITRAL GROUPS

	TOTAL GROUP	OVER AGE 45	UNDER AGE 45
Control	85.7 ± .91	88.9 ± 1.09	80.3 ± 2.3
Mitral	84.4 ± 1.35	86.2 ± 1.75	81.5 ± 2.03
Difference	1.3	2.7	1.2
S. D. difference	1.68	2.05	2.3
Critical, ratio	.75	1.32	.52

A comparison of the mitral groups with their corresponding controls on a basis of average systolic and diastolic pressures (Tables II and III) likewise results in small differences which are not statistically significant. The highest critical ratio among these is 1.32. Here, too, the observed, slight difference for the subgroup over the age of 45 years is actually the opposite of that suggested. For the mitral group, the average blood pressure was 144.4 ± 3.02, systolic, and 86.2 ± 1.75, diastolic, as compared to the control values of 148.2 ± 2.34 and 88.9 ± 1.09.

An additional check upon the control group, averages of systolic and diastolic pressures, weighted for age and sex to correspond with the control group employed, were calculated from the values given by Wetherby.[5] These values were ascertained from a study of 5,540 persons of the same population from which the control group for the present study was selected. The averages thus calculated are 143, systolic, and 86, diastolic, and compare closely with the 139.86, systolic, and 85.7, diastolic, found in the present study. It does not appear therefore, that any considerable selective factor was operative in determining the present control group.

Finally, the mitral group of 144 cases was divided into those with hypertension and those without, and the average age at death of these two groups was compared. The average age at death for those with hypertension was 56.5 ± 2.19, and for those without, 48.9 ± 2.58. The difference of 7.6 ± 2.55 may be considered as bordering on statistical significance. However, it is not safe to conclude from this that the presence of hypertension confers an increased life expectancy upon a person with mitral disease. Hypertension and age are not independent variables; the former occurs with increasing frequency at higher ages, and, for that reason, the selection of a group on a basis of hypertension entails the artificial selection of those in the higher age range.

The objection may be raised that the blood pressures recorded for the autopsy group of cases of mitral stenosis are spuriously low because

they were taken after the onset of heart failure in most cases, and on hospitalized patients, whereas those for the control group were taken largely on ambulatory patients. An attempt was made to avoid the terminal fall in blood pressure by excluding those cases in which the recording was made within twelve hours of death. Furthermore, a fall in blood pressure with the onset of heart failure with mitral stenosis does not appear to be the rule. In considering this question, Fishberg[6] says, "Sometimes the systolic and pulse pressure fall slightly. More often the onset of heart failure in mitral stenosis is accompanied by a rise in the diastolic pressure with a smaller increase in systolic pressure." He also reports, "Lang and Manswetowa found that as a rule a break in compensation in cardiac disease is accompanied by a rise in arterial pressure which falls again as the heart improves. This was especially often the case in mitral disease and emphysema heart."

Finally, it seems unlikely that false readings due to the patients' condition would, in a group of this size, so exactly counterbalance an actual difference as to yield the observed close agreement.

CONCLUSIONS

It is somewhat hazardous to draw sweeping conclusions from small samples, however rigorous statistical precautions may be exercised. It appears, however, that the close agreements which were found in the present study between mitral and control groups by all methods of comparison justify at least a skeptical attitude toward the assertion that there is a higher frequency of hypertension among persons with mitral stenosis than obtains in the general population. From the evidence presented, nothing can be concluded concerning the effect of hypertension upon the clinical course of mitral stenosis.

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CONGENITAL SUBAORTIC STENOSIS

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THE infrequency of subaortic stenosis is demonstrated by the fact that, since the first report of Chevers,[1] one hundred years ago, only thirty-two cases have been recorded. Our only knowledge of the incidence of this congenital anomaly is based on Maude Abbott's[2] series of 1,000 post-mortem cases of congenital heart disease, in which there were twelve (1.2 per cent) instances of true subaortic (infundibular) stenosis. Inasmuch as no reports on the incidence of this cardiac lesion among the population at large have been presented, the following series of ten cases, discovered during the course of examining approximately 18,000 soldiers, is of interest. Although this results in an incidence of approximately .05 per cent, the frequency of occurrence among the general population is probably somewhat higher, for a certain number of such cases were undoubtedly recognized at various induction centers, and the men consequently rejected for military service. In this respect, therefore, the men examined constitute a selected group.

PATHOLOGY

Grossly, the lesion appears as a firm, raised, fibrous ring of tissue from 1 to 15 mm. below the base of the aortic valve, usually from 2 to 4 mm. in height, and extending 1 to 10 mm. into the ventricular cavity. On microscopic examination, it is seen that this raised band, or shelf, is hyalinized connective tissue covered by intact, flattened endothelium. With Weigert's stain, the fibrous tissue has been shown by Wiglesworth[3] to consist principally of elastic fibers.

Abbott[4] attempted to classify these cases etiologically into two groups, namely, those cases in which the anomaly resulted from a congenital arrest of development, and those in which an early postnatal or late prenatal inflammatory process was considered to be the cause. A mechanical origin has also been postulated,[5] with the belief that the subaortic shelf is produced by the impact of the bloodstream at this area. The most acceptable explanation of the pathogenesis of this lesion is that of Keith,[6] who stated that the raised band represents a remnant or "arrest of atrophy" of the bulbus cordis. Wiglesworth[3] has pointed out that the participation of the endocardial elastica in the formation of the ring corroborates the concept of a congenital origin, and renders doubtful any possibility of an inflammatory or mechanical cause. Walsh, Connerty, and White[7] also think that the lesion is congenital.

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CLINICAL DIAGNOSIS

Clinically, the diagnosis of congenital subaortic stenosis is not a difficult one. The patient usually has no symptoms referable to the heart, but auscultation and palpation reveal the murmur and thrill of aortic stenosis, with a normal or nearly normal aortic second sound; the sound is not affected because the lesion does not involve the aortic cusps, but is situated below them. The blood pressure and pulse pressure are normal, and, consequently, the anacrotic pulse of aortic stenosis is not present. With these observations, in the absence of a history of rheumatic fever or suggestive rheumatic attacks in a young patient, the possibility should be suspected. As has been pointed out by Walsh, Connerty, and White,[7] the younger the patient, the more likely will the diagnosis be correct.

If other cardiac defects, congenital or acquired, should be present in addition to the subaortic stenosis, the diagnosis will undoubtedly be more difficult and uncertain. Only two cases of subaortic stenosis with other congenital cardiac defects have been reported,[7, 8] both with bicuspid aortic valves. In only one of these,[7] however, was an aortic diastolic murmur, in addition to the aortic systolic, audible. Apparently, the authors believed that this resulted from the marked degree of deformity of the valve, for, ordinarily, an isolated bicuspid aortic valve produces no auscultatory phenomena.

An unusual number of cases of subaortic stenosis with mitral stenosis have been reported by the French writers.[9-12] These, however, apparently were always of inflammatory origin, probably rheumatic, in which a "fish mouth" mitral valve was accompanied either by an extension of the inflammatory process from the mitral ring to the subaortic endocardium, resulting in the formation of a subaortic shelf (Vulpian, Liouville, and Rendu), or a thickened aortic leaflet of the mitral valve projected below the aortic valve into the ventricular cavity (Lemierre and Bernard). In all cases, the lesion was recognized at post-mortem examination only; the clinical diagnosis had been mitral stenosis, with or without aortic stenosis.

MATERIAL

All the cases were in apparently healthy young men, ranging from 19 to 30 years of age. A careful history revealed no previous attacks of rheumatic fever, epistaxis, frequent sore throats, "growing pains," or erythema. There was no history of syphilis, and serologic tests were negative. Except for two patients who complained of mild to moderate exertional dyspnea (Cases 6 and 7), no cardiac symptoms had ever been present. All had been in the army for periods of two to twelve months, during which time they had been able to perform their duties without difficulty. Only one man had any knowledge of a "heart murmur"

TABLE I

CLINICAL DATA IN TEN CASES OF CONGENITAL SUBAORTIC STENOSIS

CASE NUMBER	AGE (YRS.)	HISTORY OF KNOWN HEART DISEASE	SYMPTOMS	CARDIAC ENLARGEMENT	AORTIC AREA			CAROTIDS		BLOOD PRESSURE
					SYSTOLIC MURMUR	SYSTOLIC THRILL	SECOND SOUND	SYSTOLIC MURMUR	SYSTOLIC THRILL	
1	25	0	0	0	Harsh, rough	0	Normal	Harsh, rough	Slight	118/72
2	30	0	0	0	Moderately loud, rough	0	Normal	Loud, rough	Moderate	115/70
3	21	Known congenital heart disease	0	0	Loud, rough	Slight	Slightly decreased	Loud, rough	Marked	134/82
4	20	0	0	0	Loud, rough	0	Slightly decreased	Louder, rougher	Slight	98/68
5	19	0	0	0	Loud, rough	Slight	Normal	Louder, rougher	Marked	125/78
6	26	0	Dyspnea on exertion	0	Loud, rough	Moderate	Normal	Louder, rougher	Marked	125/84
7	25	0	Dyspnea on exertion	0	Moderately loud, rough	0	Normal	Loud, rough	Moderate	120/82
8	21	0	0	0	Loud, rough	0	Normal	Louder, rougher	Moderate	116/65
9	29	0	0	0	Moderately harsh	0	Normal	Louder, rougher	Slight	130/84
10	20	0	0	0	Moderately loud, rough	0	Slightly decreased	Louder, rougher	Moderate	116/65

prior to the present examination. He had been examined by his private physician before induction, and had been told at that time that he had congenital heart disease.

Clinical examination of these men showed a striking similarity in all; the signs were always consistent with the diagnosis of congenital subaortic stenosis. A rough systolic murmur, frequently accompanied by a systolic thrill, was present in the aortic area. On recumbency, both murmur and thrill diminished in intensity, and became more pronounced after full expiration with the patient in the upright position. The murmur was transmitted upward to the carotids, where it usually became louder and rougher. There was minimal transmission of the murmur down along the sternal borders, and in no case was there transmission to the apex or posteriorly to the bases of the lungs. In cases in which no thrill was detectable over the aortic area there was a systolic thrill of slight to moderate intensity and duration over the right carotid artery. In those in which a thrill was palpable over the second intercostal space to the right of the sternum, the intensity and duration were greatly increased in the neck. The aortic first sound was moderately obscured by the murmur, but the aortic second sound was clearly audible, varying from normal to a slight diminution in intensity.

In no case were there murmurs elsewhere, nor was there any other evidence of additional valvular involvement. Regular sinus rhythm was present in all. Unfortunately, radiologic studies could not be made, but, clinically, none of the men showed evidence of cardiac enlargement as ascertained by percussion and the location and thrust of the apical impulse. As would be expected with this type of congenital lesion, the blood pressure and pulse were normal. There was no peripheral clubbing or cyanosis, or evidence of congestive failure.

DISCUSSION

Inasmuch as post-mortem studies have not been made in any of the cases in this series, some doubt may be entertained concerning the validity of the clinical diagnosis. The only other possibility would be acquired aortic stenosis of rheumatic or sclerotic origin. In view of the negative history for rheumatic attacks, the infrequency of isolated rheumatic aortic stenosis at this age, and the absence of other valvular lesions and cardiac enlargement, a rheumatic origin is hardly likely. The age range of the patients is also strongly against the possibility of nonrheumatic, calcific, or sclerotic aortic stenosis. Furthermore, the physical signs (presence of the aortic second sound, normal pulse and blood pressure, Table I) indicate a lesion that does not involve the aortic valve, but must be located proximal to it.

Although this congenital lesion may be compatible with longevity, as evidenced by one of Sternberg's[5] patients, who lived to the age of 77 years, the prognosis in the majority of cases is poor. There is a marked tendency toward the development of bacterial endocarditis,

acute or subacute, at some time within the first three decades of life.[2, 3, 7, 8, 13, 14] Sudden death, with no pathologic changes other than the subaortic ring, has also been reported.[2, 15] In view of this prognosis, accuracy of diagnosis and the rejection of such men for military service become of paramount importance.

SUMMARY AND CONCLUSIONS

Ten cases of congenital subaortic stenosis, discovered during routine examination of approximately 18,000 soldiers, are described; this is an incidence of .05 per cent. Since the prognosis is poor, in that most such patients eventually develop bacterial endocarditis or die suddenly, persons with this disease should be rejected for military service.

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VARICES OF THE BRONCHIAL VEINS AS A SOURCE OF HEMOPTYSIS IN MITRAL STENOSIS

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HEMOPTYSIS, as a complication of rheumatic heart disease, has several unusual features. Stewart[1] defined these clearly. He stressed the blood-streaked sputum, which frequently resembles pure blood. The hemorrhage may exceed 500 c.c. Pulmonary infarction and signs of congestive heart failure are minimal or absent. The hemoptysis may accompany acute exertion, an upper respiratory infection, or auricular fibrillation. All investigators agree that the intrapulmonary blood pressure is elevated. A satisfactory source for the hemoptysis, which would explain all these features, has not been found.

Communications between the pulmonary veins and bronchial veins have been described.[2] Miller[3] demonstrated these communications by injection experiments. We decided to investigate the condition of these communications in cases of mitral stenosis in order to see whether submucous varices were present. Since the bronchial veins are difficult to locate and inject, the injections were made into the pulmonary veins with particulate matter too coarse to enter the capillaries.

PROCEDURE

Lungs fresh from autopsies were injected. The lungs were stored in an icebox overnight to permit loss of rigor, then warmed in an approximately isotonic saline solution to 40° C. before injection. A cannula was inserted into the pulmonary vein from the lower lobe, through which 500 c.c. of warm saline solution were forced to wash out the blood. The injection mass was then introduced through the cannula at a pressure of 80 mm. Hg. This mass was a modification of that originally suggested by Schlesinger[4] for heart injections, and consisted of:

White lead	300 Gm.
Cinnabar	50 Gm.
Sucrose	120 Gm.
Gelatin	70 Gm.
Water	1,000 c.c.

This mixture was fluid at 40° C. The injection pressure was maintained several minutes after immersing the lung in ice water, and then the injected lung was returned to cold storage to allow the mass to harden. Roentgenograms were taken of the first few preparations to check on the completeness of the injection technique, which was found to be satisfactory.

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CONTROL CASES

There were seven control cases without clinical signs or pathologic evidence of mitral stenosis. There were six females and one male, aged 26, 39, 47, 59, 59, 62, and 71 years. Three of the patients had hypertension, but only one had terminal cardiac failure in association with uremia. The youngest control was the case of a woman, 26 years of age, who died of a self-administered overdose of barbiturates. The lung showed the same degree of development of bronchopulmonary venous anastomoses and the same pattern of submucosal bronchial veins as the lungs of older patients who died from natural causes. All these cases exhibited similar features.

Fig. 1.

Fig. 2.

Fig. 1.—Typical large bronchus, in the gross, from a 59-year-old, normal woman who died in diabetic coma. There was generalized arteriosclerosis but no cardiac failure. No injected veins are seen, but longitudinal mucosal folds are visible. *A*, Bronchial folds.

Fig. 2.—Small bronchus from a 26-year-old, normal woman who died from an overdose of sedatives. Note the injected bronchial veins, some forming spider-like patterns and others with no definite arrangement. *A* and *B*, Spider-like pattern of the bronchial veins.

In the gross, the bronchial veins, filled with the injected mass, appeared as orange lines in the bronchial mucosa of the smaller and medium-sized bronchi. These veins were conspicuous, and either formed irregular spider-like patterns or had no regular arrangement. Few or no vessels were visible in the larger bronchi. Figs. 1 and 2 show typical large and small bronchi in the gross.

Many very small injected vessels were seen microscopically in the bronchial submucosa. The larger vessels were deeper in the submucosa and were well separated from the lumen of the bronchus. In the spaces between the cartilaginous plates there were larger and more deeply-placed vessels. These seemed to connect the bronchial and pulmonary veins. In the smaller bronchi, the submucosa was thinner and the veins were more superficially placed, making them more conspicuous in the gross (Figs. 3 and 4).

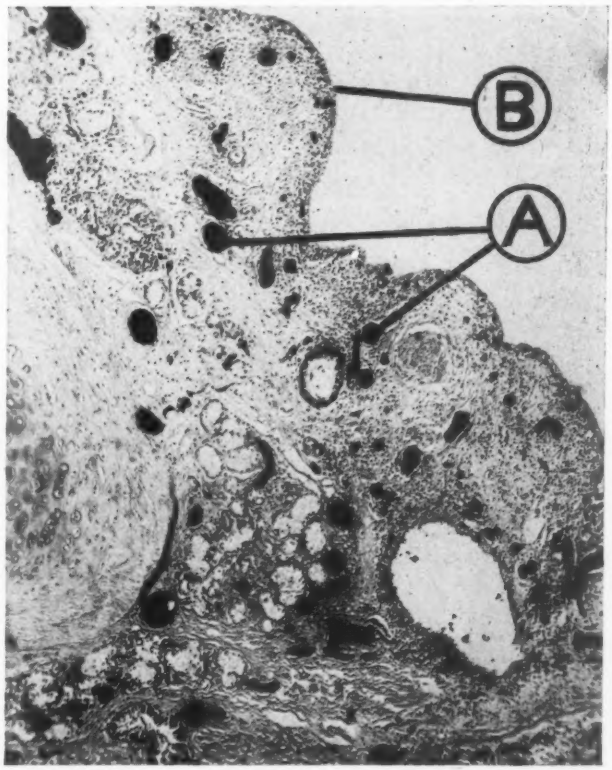

Fig. 3.—Section of a large bronchus from a 62-year-old woman, showing the injected bronchial veins (solid black areas). Note the mucosal fold often seen in the gross. (×90.) *A*, Injected bronchial veins. *B*, A mucosal fold.

The injected material was also seen in the veins of the parenchyma of the lung. It did not appear in the pulmonary arteries and capillaries, nor in the bronchial arteries and capillaries. The particles of mercuric and lead salts did not enter channels less than 20 microns in diameter.

NONRHEUMATIC CARDIAC CASES

There were six cases of cardiac failure in this group. The ages of the patients were 39, 53, 57, 58, 63, and 71 years. Four of the patients

had marked hypertension, with cardiac failure. The 58-year-old patient had syphilitic heart disease and aortic insufficiency. He died of congestive heart failure. At autopsy there was syphilitic involvement of the coronary ostia. The sixth patient, aged 63 years, an alcoholic, died of congestive failure. The heart weighed 790 grams, and there were mural thrombi in the right auricle and left ventricle. There was no evidence of coronary occlusion, and the possibility of beriberi heart disease was considered.

Fig. 4.—Section of a smaller bronchus from the same case as Fig. 3. The mucosa is thinner and the veins are more superficial, although of the same size as in the large bronchi. (×90.)

Three of these patients showed slight dilatation of the bronchial veins in the larger bronchi. The veins were definitely more conspicuous than in the normal controls. Fig. 5 demonstrates these changes. The photograph was made from the 39-year-old patient who suffered from arteriolar nephrosclerosis and hypertension. He had coughed up 60 c.c. of frank blood two days before death. The lungs showed some small scattered areas of consolidation in both lower lobes, but no areas of infarction. The bronchial mucosa was markedly hyperemic.

MITRAL STENOSIS CASES

The patients in this group had histories suggestive of rheumatic heart disease, and autopsy reports confirmed the diagnosis of mitral stenosis. Eleven such cases were studied. In six there was some evidence of dilated venous channels, in four of which there were innumerable, wide venous channels running parallel to the lumen in the secondary and tertiary bronchi and disappearing in the main bronchus. A few important details are summarized as follows:

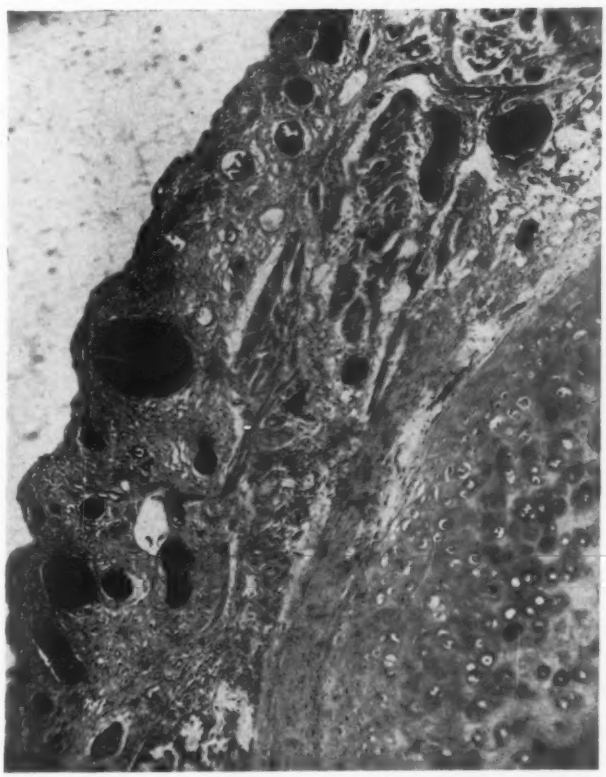

Fig. 5.—Section from a large bronchus from a 39-year-old patient who died of congestive heart failure as a result of long-standing hypertension and arteriosclerosis. Hemoptysis (60 c.c.) occurred twenty-four hours before death. The dilated bronchial veins may be seen as large black areas just beneath the surface of the bronchial mucosa.

CASE 1.—A 33-year-old woman had had chorea and joint pains at the age of 6 years. Mitral stenosis was diagnosed at that time. She was hospitalized eleven times thereafter. Auricular fibrillation had been present for fifteen years. Her first hemoptysis occurred nine years before death. On her last admission there were signs of advanced cardiac failure. Autopsy showed involvement of the mitral, tricuspid, and aortic valves. The left auricle comprised one-half the volume of the heart. The veins of the visceral pleura were dilated and anastomosed with dilated veins in the diaphragm. The injected specimen showed numerous dilated, tortuous bronchial veins in the larger bronchi.

CASE 2.—A 16-year-old girl was first found to have an enlarged heart and systolic and diastolic murmurs at the age of 7 years. Dyspnea gradually increased during the next seven years. At the age of 15 years she became acutely decompensated. The only hemoptysis occurred the day before death. Normal sinus rhythm was present. She died of cardiac failure. Autopsy revealed cardiac hypertrophy, myocarditis, mitral stenosis, and thrombi in both auricles. There was definite pulmonary arteriosclerosis, and there were thrombi in the pulmonary arteries, with infarcts. Marked varices of the bronchial veins in the larger bronchi were found on injection (Figs. 6 and 7).

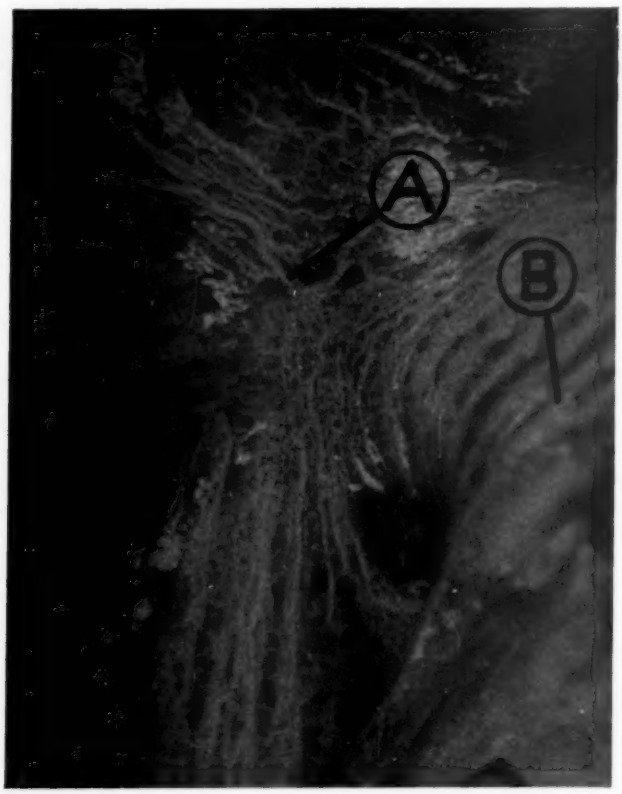

Fig. 6.—The large bronchi from a 16-year-old girl with mitral stenosis (Patient D.M.). The dilated bronchial veins are seen, along with some mucosal folds. The veins stream toward the hilum, forming a collateral circulation. *A*, Dilated bronchial veins. *B*, Mucosal fold.

CASE 3.—A 33-year-old woman had rheumatic fever in childhood. At the age of 23 years she first noticed attacks of tachycardia, and, four years later, dyspnea became pronounced. Five months before death hemoptysis occurred. There were physical signs of mitral stenosis and insufficiency and auricular fibrillation. She developed an acute terminal pneumonia. The autopsy diagnosis was mitral stenosis and insufficiency, hemorrhagic bronchitis, and pulmonary congestion. Because of the acute hemorrhagic bronchitis it was impossible to see any dilated bronchial veins in the injected specimen.

CASE 4.—This 37-year-old patient had her first attack of rheumatism at the age of 12 years. There were no cardiac symptoms until the age of 38 years, during her pregnancy, when she developed mild cardiac failure. There was no history of hemoptysis. She was admitted with signs of acute rheumatic heart disease, mitral stenosis and insufficiency, and normal sinus rhythm. Autopsy revealed a large heart and mitral stenosis and insufficiency, with calcification of the leaflets. There were a few large infarcts in the lungs. The injected specimen showed fine, linear veins in the mucosa of the larger bronchi.

Fig. 7.—Section of a large bronchus from the same case as Fig. 6. Note the great dilatation of the bronchial veins and their situation close to the lumen of the bronchus. (×90.)

CASE 5.—This girl was 27 years of age. Four years previously she was told she had heart disease. She was admitted with subacute bacterial endocarditis. There were signs of mitral stenosis and insufficiency and aortic stenosis and insufficiency, with normal sinus rhythm. Autopsy confirmed this diagnosis. There were no pulmonary infarcts. The injected specimen showed enlarged, slightly tortuous, longitudinally arranged veins near the bifurcation of the larger bronchi.

CASE 6.—This 30-year-old woman had rheumatic fever and chorea at the age of 7 years. Rheumatic heart disease was diagnosed six years before death. She died, in her eighth month of pregnancy, of acute

pulmonary edema. There was no history of hemoptysis. The heart rhythm was regular. Autopsy revealed aortic stenosis and insufficiency, with only moderate evidence of mitral stenosis. The injected lung specimen revealed only slight dilatation of the bronchial veins in the major bronchi.

CASE 7.—This 31-year-old man had attacks of rheumatic fever at the ages of 8 and 13 years. There was no history of hemoptysis. He was admitted to the hospital in congestive heart failure, with signs of both aortic and mitral valve involvement and normal rhythm. Autopsy confirmed the diagnosis of the valvular lesions. The injected specimen revealed marked injection and dilatation of the bronchial veins. The veins were numerous and tortuous, and streamed toward the hilum of the lung.

CASE 8.—This patient was a 42-year-old housewife. There was no history of rheumatic fever, but, at the age of 36 years, she was told she had heart disease. There was no history of hemoptysis. The heart was enlarged and there were signs of mitral stenosis and insufficiency and auricular fibrillation. She died of congestive heart failure, accompanied by bronchopneumonia. At autopsy the mitral valve was typically "buttonhole" in character. The injected specimen revealed no dilatation of the bronchial veins, and microscopic sections showed only moderately dilated veins deep in the bronchial mucosa.

CASE 9.—This 52-year-old woman died of pulmonary infarction and rheumatic heart disease. There was no history of hemoptysis preceding her terminal illness. Autopsy revealed definite mitral stenosis and thickening of the leaflets of the aortic valve. The injected lung specimen showed no evidence of dilated veins.

CASE 10.—This man was a 56-year-old hypertensive patient with mitral stenosis and insufficiency and auricular fibrillation. Autopsy confirmed the diagnosis of mitral stenosis, and there was also pulmonary arteriosclerosis. There was no definite dilatation of the bronchial veins in the injected specimen.

CASE 11.—This patient was 57 years old. Symptoms of decreasing cardiac reserve had been present over a period of four years. Three weeks before admission he had hemoptysis and pain in the chest. Auricular fibrillation was present, and autopsy revealed mitral stenosis and insufficiency, with a dilated left auricle. There was no evidence of pulmonary infarction. The pulmonary arteries showed marked arteriosclerosis. The injected specimen showed many anastomotic vessels in the bronchial mucosa, and the sections revealed large, dilated bronchial veins which lifted the mucosa into the lumen of the bronchus.

Four of these patients had hemoptysis at some time, but in only one case could it be correlated with pulmonary infarction. A marked hemorrhagic bronchitis was present in Case 3, and it was impossible to detect the presence of dilated veins. Six of the eleven patients showed definite evidence of enlarged bronchial veins. Four of these six patients showed greatly dilated bronchial veins along the longitudinal axis of the large bronchi. They were fairly straight and parallel, and seemed to stream toward the main bronchus. Fig. 6 shows a photograph of a large bronchus from Case 2. The large mucosal folds

are visible, as well as the innumerable dilated veins on the surface of the mucosa. Fig. 7 is a section of one of the bronchi, showing the injected bronchial veins lying just under the mucosal surface.

Fig. 8.—Section of a large bronchus from a 56-year-old man with mitral stenosis, but no history of hemoptysis (Patient G.Z.). The bronchial veins are somewhat enlarged, but not very superficial. (×90.)

DISCUSSION

Most authorities seem to agree that there are only capillary anastomoses between the pulmonary and bronchial circulations. Wiggers[5] states that there are no functional arterial or venous anastomoses. Wood, Crever, and Miller[6] and Daly[7] also state that there are only connecting capillaries on the arterial side. In another article, Berry and Daly[8] indicated the further possibility of small arteriole or venule connections. The injection mass which we employed was never seen in the pulmonary capillaries, and it did not appear in the bronchial or pulmonary arteries. The smallest vessels filled with the mass measured approximately 20 microns in diameter. However, the injected material passed freely, even in the control cases, from the pulmonary to the bronchial veins. The injection pressure used (80 mm. Hg) might have been high enough to dilate capillary anastomoses. This seems

unlikely because the injection mass did not enter the capillaries in the parenchyma of the lung. There must be innumerable venules anastomosing the bronchial and pulmonary venous beds, both in normal lungs and in cases of heart failure.

Whatever the type of anastomosis, the blood flow through it is thought to be from the bronchial to the pulmonary circulations, according to Miller,[3] Berry and Daly,[8] and others, although there are no data on normal, left auricular pressures in man. We believe that when mitral stenosis is interposed, the flow to the left auricle through the pulmonary veins is hindered, and the pressure in these veins rises above that in the right auricle. The blood flow through the anastomosis is reversed, and the blood passes from the pulmonary circulation into the bronchial veins and back to the right side of the heart, via the azygos, hemiazygos, and intercostal veins. The submucosal bronchial veins leading toward the main bronchi dilate greatly to handle this collateral flow, and even become grossly visible. The hemoptysis which often accompanies mitral stenosis, yet is not associated with acute pulmonary edema or pulmonary infarction, is probably caused by the rupture or ulceration of these engorged bronchial veins. This hemoptysis resembles massive bleeding from hemorrhoids and esophageal varices, which, like the bronchial veins, are submucous shunts between large venous drainage areas. A severe coughing spell, mild ulcerative bronchitis, or a rise in left auricular pressure could initiate such attacks of hemoptysis.

This view differs widely from the explanations previously offered. The most frequently suggested opinion was that the extreme congestion and hypertension of the pulmonary arterial and venous bed eventually caused these vessels to rupture. A long list of writers, including White,[9] McKenzie,[10] Vaquez,[11] Stewart,[1] and Ginsberg,[12] have held this opinion, which implies simply an accentuation of the diapedesis of erythrocytes into the alveoli in heart failure. Wolff and Levine[13] suggested this same cause, but felt that sclerosis of the vessels might be a necessary precursor of the hemorrhage. As Brenner[14] pointed out, pulmonary arteriosclerosis frequently does accompany mitral stenosis. However, two of our four patients with hemoptysis showed no evidence of pulmonary sclerosis. Proft, as quoted by Oppenheimer and Schwartz,[15] explained the hemoptysis as due to sudden dilatation of the lung capillaries, with diapedesis into the alveoli, or to dilatation with rupture of the capillaries lining the small bronchioles. It does not seem likely that diapedesis into the alveoli could account for the sudden profuse hemorrhages which often occur. In all of our cases of mitral stenosis there were "heart failure" cells in the alveoli. Only one of our four patients with hemoptysis had gross areas of bleeding, resembling infarcts, in the lung parenchyma. The vessels of the smallest bronchi and bronchioles did not differ from the normal. For this

reason it does not seem likely that the bleeding arises from the small vessels in the bronchiolar system.

Estimates of the actual incidence of pulmonary hemorrhage in mitral stenosis range from Wolff and Levine's 10 per cent[13] to Hay and Hunt's 23 per cent.[16] Hemoptysis in mitral stenosis has long been recognized as a poor prognostic sign (Wolff and Levine[13]). The bleeding, per se, is not dangerous. However, it is the result of long-standing and marked mitral stenosis. Hence, the hemorrhage indicates a very poor cardiac status and is correlated with a poor prognosis. Our four patients with hemoptysis lived one day, four weeks, three months, and nine years, respectively, after their initial pulmonary hemorrhage. The patient (Case 1) who lived nine years had the most marked evidence of collateral bronchial venous flow, and a collateral circulation had also been set up through the veins of the diaphragm.

Age has no effect upon the normal capacity of the bronchial veins. Our seven controls ranged in age from 26 to 71, and there were no observable differences in the size of their vessels. The oldest of our mitral stenosis patients was 57, and the youngest, 16 years. Hemoptysis occurred in both of these and in two other patients at the age of 33 years, so that this bleeding shows no age relationships. Hypertension also does not appear to affect the normal capacity of the bronchial veins unless chronic congestive heart failure occurs. There were no changes in the bronchial veins in any of the normal controls, and coronary and pulmonary arteriosclerosis did not affect the flow through the bronchial veins when it occurred in the patients.

SUMMARY

1. A method for injecting the bronchial veins is described.

2. This method indicates the presence of direct venous connections between the bronchial and pulmonary veins in men of all ages.

3. Mitral stenosis causes dilatation of the bronchial veins in the submucosa of the larger bronchi as a result of the establishment of a collateral flow through them.

4. In cases of mitral stenosis in which infarction and acute pulmonary edema are not present, hemoptysis is probably due to bleeding from these dilated veins.

5. Age, hypertension, and arteriosclerosis do not affect the bronchial venous bed, but some dilatation occurs in chronic congestive heart failure of long standing, although the only lesion of the mitral valve may be dilatation of the valve ring.

We are indebted to Doctor Jacob Werne for some of the autopsy material, and to Doctor William Dock for the technique and for valuable material and advice.

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THE ASSOCIATION OF PAROXYSMAL ATRIAL TACHYCARDIA WITH ATRIAL FLUTTER OR FIBRILLATION

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IN A RECENT study of our cases of paroxysmal atrial tachycardia in which A-V block was present,[1] we were surprised to note the not infrequent association of paroxysmal tachycardia with atrial flutter or fibrillation. Although it is well recognized that flutter and fibrillation are frequently associated, and probably are manifestations of mechanisms which are fundamentally the same, it has been generally felt that paroxysmal atrial tachycardia is different, specifically and generically, and bears no relationship whatsoever to a circus mechanism. Without attempting to discuss the fundamental nature of any of these three disturbances of the cardiac mechanism, we wish to show in this communication that this type of paroxysmal tachycardia with A-V block is more intimately associated with flutter and/or fibrillation than has hitherto been appreciated.

Existing evidence on this point is scanty. Hirschfelder,[2] in experiments employing faradism of the auricular appendage of the dog, found that stimuli of low or moderate intensity gave rise to paroxysmal tachycardia, whereas intense and prolonged stimulation produced auricular fibrillation. This led him to conclude that "true paroxysmal tachycardia is usually caused by increased irritability of the heart muscle, especially of the auricles, which probably pass into a state of fibrillation." This possibility was denied by Lewis,[3] although he described in great detail a case[4] in which a short period of fibrillation was recorded during an atrial paroxysm. He also noted the fact, which must have been apparent to many, that atrial premature beats commonly precede the ultimate appearance of either paroxysmal tachycardia or fibrillation. Also, in experimental animals, rapid, rhythmic stimulation of the auricle simulated tachycardia, whereas increased rates of stimulation produced flutter or fibrillation.[5]

In 1932, Carr[6] reported a unique instance of paroxysmal tachycardia, apparently of A-V nodal origin, in which short periods of atrial flutter interrupted the paroxysms. On a single occasion, pressure on the right carotid sheath resulted in a shift from paroxysmal tachycardia to flutter with high-grade A-V block. After the administration of $\frac{1}{100}$ grain of atropine subcutaneously, the tachycardia changed within one minute to

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flutter; this was soon followed by sinus rhythm with frequent atrial premature beats, and short atrial paroxysms appeared later.

Brown[7] has also recorded a single case in which paroxysmal atrial tachycardia was closely associated with a circus movement. His patient was overdigitalized, and, as a result, A-V block appeared, and the mechanism was shortly converted to fibrillation. Because of the A-V block, the electrocardiograms had been thought by some to represent atrial flutter rather than tachycardia, and Brown resorted to esophageal leads to make the differentiation clear.

Recently, Barker, et al.,[8] have described eighteen examples of paroxysmal tachycardia with A-V block. Among this group there were three patients who also showed flutter or fibrillation. They state that, of 100 cases of paroxysmal atrial tachycardia, in only five was the patient known to have had flutter or fibrillation also; in three of the five, partial A-V block was present during the tachycardia. In their Case 2, flutter was first seen; four days later atrial tachycardia was recorded, and it is worth noting that, in the interim, 0.85 Gm. of digitalis had been administered intravenously. The further injection of 0.35 Gm. of digitalis produced 2:1 A-V block. Digitalis was continued by mouth, and the tachycardia with block persisted. Two months later there was normal sinus rhythm, and this appeared to persist. After ten months more, an electrocardiogram showed flutter in the first two leads and fibrillation in the third lead.

In Case 10 of Barker, et al., atrial tachycardia with A-V block was thought to have been due to excessive digitalization. Two days later atrial fibrillation was recorded. Case 12 showed a similar transition. This patient had been taking digitalis for three years. The day after admission he was given an additional 0.5 Gm., and the tracing taken the following day showed atrial paroxysmal tachycardia with A-V block. Two days later atrial fibrillation was present. The apparent role played by overdigitalization in these three cases, both in producing the tachycardia and in producing block, agrees with our own experience[1] in this connection.

Two additional cases have been reported which have been interpreted by the authors as illustrating the conversion of paroxysmal tachycardia to flutter. It is necessary at this point to recall the fact that there appears to be only one clear criterion for the differentiation of tachycardia and flutter, i.e., the presence or absence of a continuous atrial circus mechanism, as indicated by the absence or presence of an isoelectric period in the electrocardiogram. When an isoelectric period is found it indicates that continuous electrical activity of the atrium, due to a circus movement, is not present. Parkinson and Mathias[9] have recorded a remarkable case of paroxysmal tachycardia in which there was a cyclic variation in the atrial rate. As the rate rose from 155 to about 225 per minute, A-V block obviously developed. The authors

took this to mean that flutter had replaced the tachycardia, but our own opinion of the tracings would indicate merely that A-V block had developed during the time when the atrial rate was most rapid. We have reported similar cases.[1]

The impression has hitherto been current that A-V block was in itself prima-facie evidence that flutter was present rather than tachycardia. The paper of Barker, et al.,[8] and our report[1] should dispel this impression. Geraudel[10] has apparently fallen into this same error, and has gone so far as to deny the very existence of paroxysmal tachycardia, and to say that all such cases are flutter with 1:1 A-V conduction.

MATERIAL

Most of the cases cited were found during our previous study of A-V block in tachycardia. A few were not included at that time because of uncertainty as to their true interpretation. When we noted the high incidence of flutter or fibrillation among cases of tachycardia with block, we confidently expected to discover many more examples of such association among the more numerous cases of tachycardia without A-V block. In this anticipation we were disappointed; only two additional cases were forthcoming, thus amply confirming the similar experience of Barker and his associates. In one of them, the association was not intimate (Case 1); in the other (Case 13), fibrillation immediately followed the termination of a typical attack of tachycardia. Tabulation (Table I) of all of our cases shows that flutter, or fibrillation, is ten times more likely to be found in those cases of paroxysmal tachycardia in which there is A-V block than it is when such block is absent.

TABLE I

CASES OF SUPRAVENTRICULAR PAROXYSMAL TACHYCARDIA

ATRIOVENTRICULAR BLOCK		ASSOCIATION WITH FLUTTER OR FIBRILLATION	
		PRESENT	ABSENT
Present	42 cases	12 (28.6%)	30
Absent	70	2 (2.86%)	68
	112	14	98

CASE REPORTS

CASE 1.—J. R., a white woman, aged 33 years, John Sealy Hospital No. 19452, had been under observation since 1926 because of rheumatic heart disease with mitral stenosis. Palpitation and precordial pain had been the chief symptoms of cardiac origin. The first electrocardiogram taken on this patient was during an attack of paroxysmal tachycardia on Sept. 6, 1932 (Fig. 1, *A*). The next day sinus rhythm was present, with a normal P wave. She was admitted to the hospital again in October, 1932, and was found to have acute fibrinous pericarditis. Frequent atrial premature beats were noted in many tracings. Digitalization was begun on Dec. 13, and stopped on Jan. 15, 1933. On January 17, she was observed in a paroxysm of

tachycardia, rate 160 per minute, which was stopped by carotid sinus pressure; the rate fell immediately to 60 per minute. On January 19, atrial fibrillation was recorded (Fig. 1, *B*). Again, on January 21, a paroxysm of tachycardia was stopped by pressure over the carotid sinus; on January 23 she was fibrillating again. During several succeeding years there were numerous atrial premature beats. In April, 1938, hydrops of the gall bladder was drained; postoperatively, fibrillation appeared, and was controlled by digitalis. After the withdrawal of digitalis the sinus pacemaker spontaneously resumed its role. Later in 1938, during an attack of bronchopneumonia, atrial fibrillation was present for a few days, but disappeared spontaneously.

CASE 2.—A. D., a white girl, aged 14 years, John Sealy Hospital No. 66298, entered the hospital April 17, 1940. For four years she had had attacks of dyspnea associated with tachycardia, vertigo, and pain in the chest. The heart rate was found to be 188 per minute; the blood pressure was 78/56. Quinidine was given on April 19 and again on April 21. On April 24, digitalization was begun, and by April 28 the dosage was reduced to 1½ grains daily, at which level it was held during the remainder of her stay in the hospital. Electrocardiograms taken during the ten days after admission showed gradual slowing of the heart rate, but with persistence of the abnormal P wave (Fig. 2, *A*). In spite of the atrial slowing, partial A-V block with dropped beats was noted first on April 26, and was found in tracings taken at intervals until May 2. On May 16, an attack of tachycardia developed, with a heart rate of 250 per minute. Carotid sinus pressure, ocular pressure, vomiting induced by ipecac, acetyl-β-methylcholine, and quinidine were used, but produced only transient slowing of the heart, and death occurred late that day. We cannot be certain that this final attack was one of atrial flutter (Fig. 2, *B*), but the case is included because this seems to be a likely electrocardiographic, as well as clinical, diagnosis, for the bizarre P waves were no longer present, and carotid sinus pressure produced transient slowing. Unfortunately, no curves were obtained during any of the brief periods of cardiac slowing.

Necropsy showed an enlarged heart, weighing 530 grams. The ductus arteriosus was patent, and there was congenital hypoplasia of the aorta.

CASE 3.—K. L., a white man, aged 45 years, John Sealy Hospital No. 57248, was admitted March 7, 1939, in congestive heart failure; the blood pressure was 252/168. Lumbar sympathectomy had been performed fourteen months previously in an effort to relieve this marked hypertension, but no benefit had resulted. In the five days before this admission, the patient had taken 15 tablets of digitalis leaf, each of 1½ grains. On March 13, before 2.5 mg. of digoxin were given intravenously, sinus tachycardia was present; the next day, atrial fibrillation was recorded (Fig. 3, *A*). On March 17, sinus rhythm was present again (Fig. 3, *B*). On that day, and again on March 18 and 19, 3 grains of digitalis were given each day. On March 20, paroxysmal atrial tachycardia was present, with partial A-V block (Fig. 3, *C*). Digitalis was given again two days later, and the paroxysmal tachycardia recurred; it was still present on March 27.

During this period renal insufficiency had become progressively more severe, and the patient died in uremia on March 29. At necropsy the heart weighed 670 grams; there were marked nephrosclerosis and a few atheromatous plaques in the aorta.

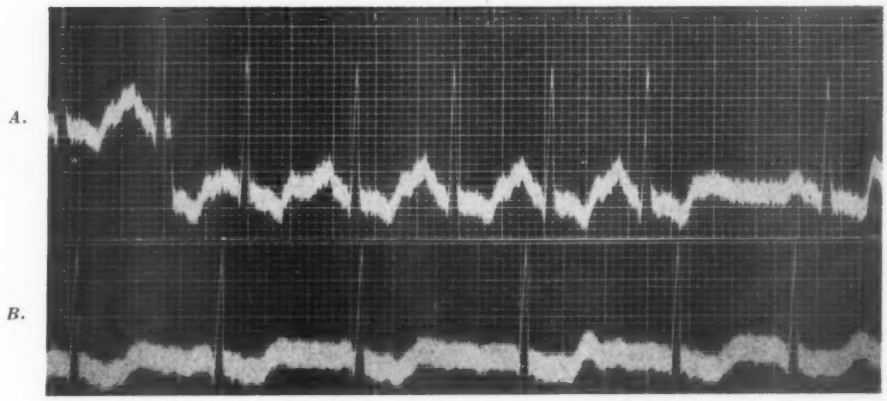

Fig. 1.—Case 1. *A*, Lead II; the termination of a paroxysm of atrial tachycardia recorded on Sept. 6, 1932. *B*, Lead II; atrial fibrillation, Jan. 19, 1933.

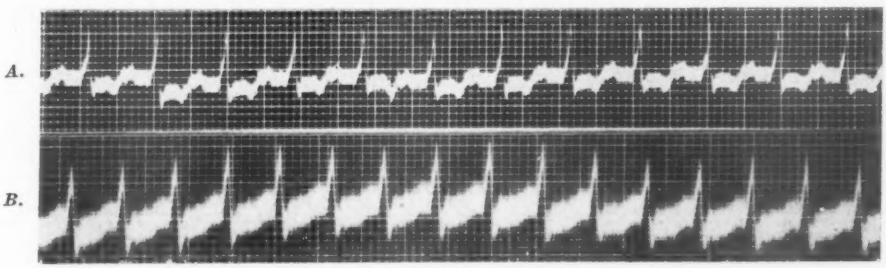

Fig. 2.—Case 2. *A*, Lead II; paroxysmal atrial tachycardia, April 17, 1940. *B*, Lead II; taken during terminal attack of tachycardia, May 16, 1940; probably atrial flutter.

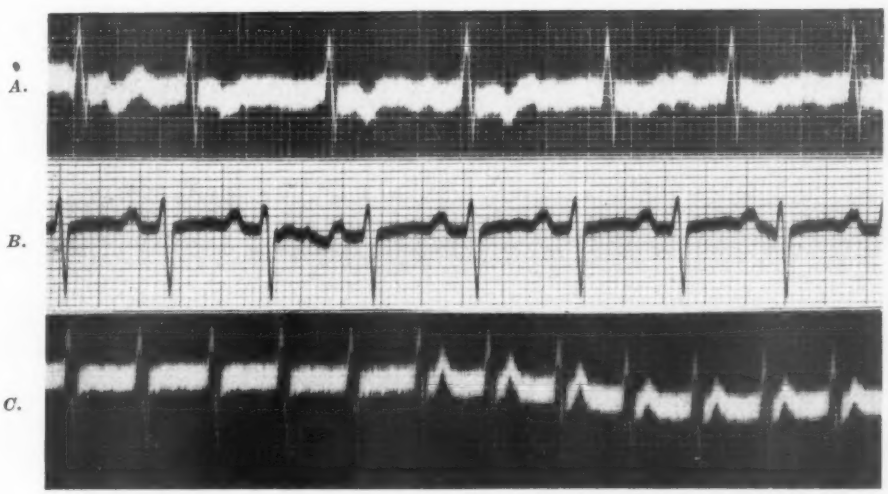

Fig. 3.—Case 3. *A*, Lead II; showing atrial fibrillation on March 14, 1939. *B*, Lead II; sinus rhythm on March 17. *C*, Lead II on March 20; paroxysmal atrial tachycardia; partial A-V block was apparent in parts of the curve.

CASE 4.—T. R., a white man, aged 49 years, John Sealy Hospital No. 66787, entered the hospital Oct. 15, 1940. He had been seen on several previous occasions because of hypertensive heart disease with congestive heart failure, and on this admission he presented the usual manifestations of this condition.

In the period from October 22 to December 1, he received 3.2 Gm. of digitalis leaf. On December 1 he was given 2 c.c. of mercupurin intravenously. Atrial fibrillation was present the next day (Fig. 4, *A*), but was replaced by sinus rhythm after 18 grains of quinidine. The following day, December 3, paroxysmal atrial tachycardia was found, at a rate of 182 per minute, with partial A-V block (Fig. 4, *B*). The ectopic pacemaker still controlled the atria on December 4, although the rate had dropped to 135 per minute. Sinus rhythm then reappeared.

Death occurred on December 14. Necropsy showed a heart weighing 600 grams, with only slight coronary arteriosclerosis. Ante-mortem thrombi were present in the right atrium and in both ventricles, and there were multiple pulmonary infarcts. There were the usual evidences of nephrosclerosis and chronic passive congestion.

CASE 5.—E. S., a colored woman, aged 68 years, John Sealy Hospital No. 75227, had been hospitalized in March and again in August, 1942, for hypertensive and arteriosclerotic heart disease in the stage of congestive heart failure. The electrocardiograms taken during this time showed numerous supraventricular premature beats, and the first tracing recorded during the August admission showed atrial fibrillation (Fig. 5, *A*); subsequent ones at this time showed sinus rhythm.

Her last admission was on March 25, 1943, when she had a recurrence of severe congestive heart failure in spite of the fact that for the preceding three weeks she had been taking 0.3 Gm. of digitalis leaf daily. Nausea and vomiting had appeared before entrance to the hospital. The electrocardiograms taken March 26 showed a basic sinus rhythm, but with many short runs of atrial premature beats (Fig. 5, *B*). On March 29 the pulse rate was counted at 170 per minute. The electrocardiograms taken that day showed atrial flutter with 2:1 A-V block (Fig. 5, *C*); this same mechanism was found again on March 31. On this day, two 5-grain doses of quinidine were given. On April 1 the flutter had disappeared, and there was again a sinus rhythm with frequent short runs of atrial premature beats; six doses of quinidine were given, but the numerous extrasystoles persisted. During the remainder of her stay in the hospital she was given 5-grain doses of quinidine three times daily. The short paroxysms of atrial tachycardia (Fig. 5, *D*) persisted until her death, April 18. The rate of the ectopic focus was highly variable; occasionally partial A-V block appeared, with dropped ventricular beats.

CASE 6.—F. G., a colored man, aged 50 years, John Sealy Hospital No. 10629, was admitted Jan. 20, 1942. Hypertensive and arteriosclerotic heart disease had been diagnosed twelve years previously, and for six years exertion had caused dyspnea. During a previous admission in October, 1941, atrial fibrillation had been recorded (Fig. 6, *A*). During the intervening months he had experienced repeated attacks of paroxysmal tachycardia, associated with dyspnea and precordial pain. In the three months preceding the present admission, the patient had taken 0.2 Gm. of digitalis leaf daily. The electrocardiogram taken January 20 showed paroxysmal atrial tachycardia with an atrial rate of 214 per minute. The next day the rate was 200 per minute, with 2:1

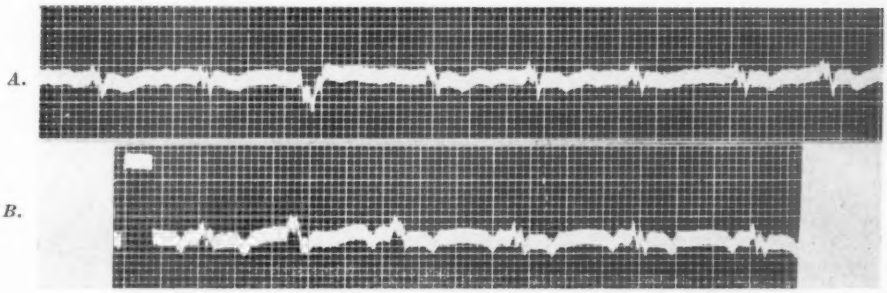

Fig. 4.—Case 4. *A*, Lead II on Dec. 2, 1940; atrial fibrillation. *B*, Lead II on December 3; showing paroxysmal tachycardia with partial A-V block.

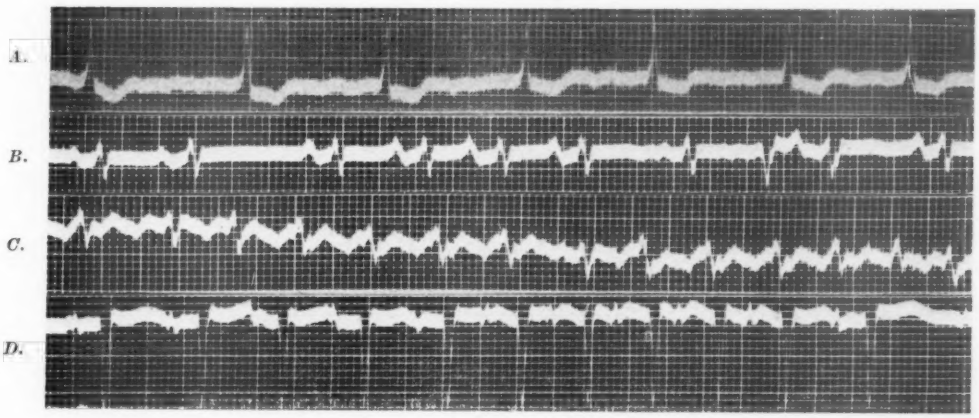

Fig. 5.—Case 5. *A*, Lead I on Aug. 4, 1942; atrial fibrillation. *B*, Lead II on March 26, 1943; showing short runs of atrial premature beats. *C*, March 29, Lead II; atrial flutter. *D*, Lead CF_2 on April 6, 1943; showing a short paroxysm of tachycardia.

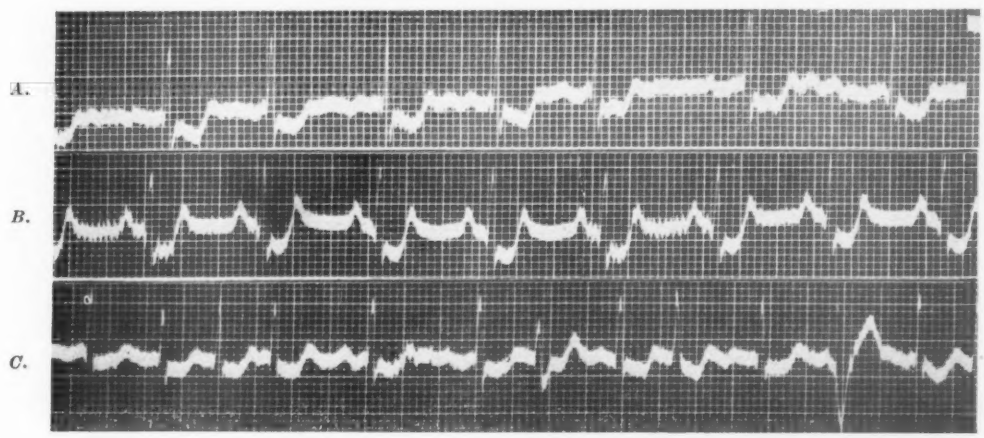

Fig. 6.—Case 6. *A*, Lead II on Oct. 24, 1941; auricular fibrillation. *B*, Lead II, Jan. 21, 1942; paroxysmal atrial tachycardia, with 2:1 A-V block. *C*, Lead II on Oct. 15, 1942; showing a return of fibrillation.

A-V block (Fig. 6, *B*). When the patient sat up there was 1:1 conduction; when he lay down, or when pressure was applied to the carotid sinus, partial block, varying from 2:1 to 4:1, appeared. He was seen again as a clinic patient in October, 1942. At this time atrial fibrillation was again present (Fig. 6, *C*).

CASE 7.—E. V., a white woman, aged 33 years, John Sealy Hospital No. 68211, entered the hospital Oct. 31, 1941. She presented a syndrome characterized by hypertension (174/130), 4 plus proteinuria, generalized edema, pulmonary infarction, and myocardial failure. In June, 1940, she had had a cesarean section, followed by pneumonia and thrombophlebitis. Since then there had frequently been edema of the right leg. Two months before admission she was delivered of a 7½-month-old fetus without complication, although during the pregnancy she had had edema of the ankles and swelling of the eyes. She had orthopnea, a continual cough, and a small amount of pink, frothy sputum.

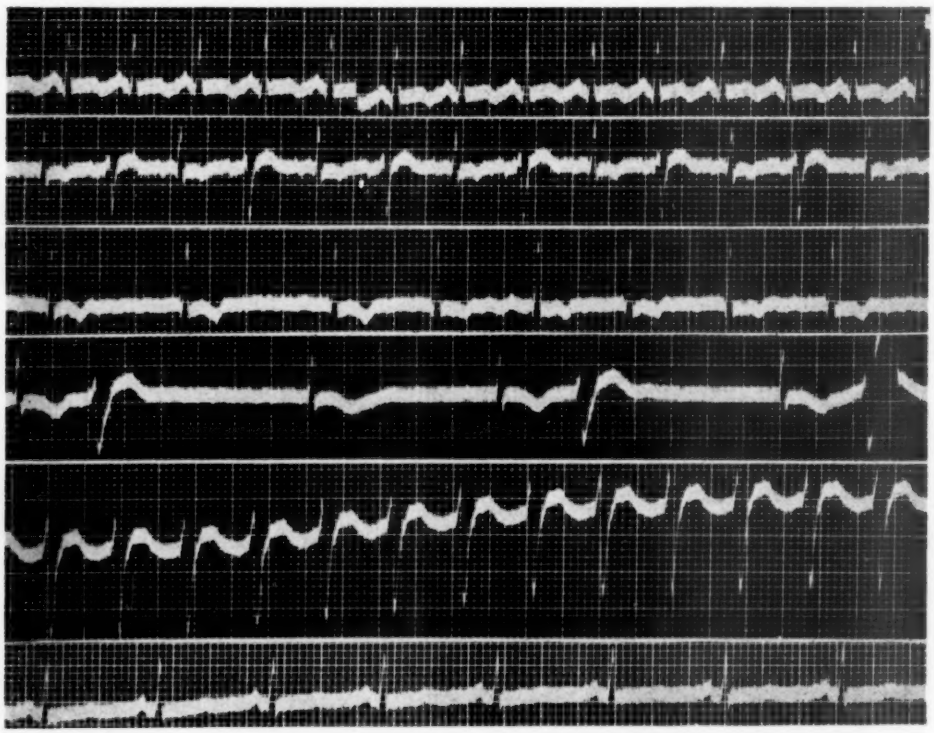

Fig. 7.—Case 7. *A*, Lead I, Oct. 31, 1941; paroxysmal atrial tachycardia. *B*, Lead I, November 4; showing alternation. *C*, Lead I, November 5; atrial fibrillation. *D*, November 11, Lead I; showing persistence of fibrillation. *E*, Lead IVF, also on November 11; showing a short paroxysm of tachycardia. *F*, Sinus rhythm on November 24; Lead I.

The urine showed marked proteinuria, a specific gravity of 1.030, many leucocytes, and a few hyaline and granular casts; erythrocytes were rare. Electrocardiograms taken on the day of admission showed supraventricular tachycardia with an atrial rate of 171 per minute (Fig. 7, *A*). On October 31, 1.5 mg. of digoxin were given, and on the next day 0.5 mg. more was administered. On that day, November 1, the atrial

rate dropped to 136, but the focus was still an ectopic one. From November 1 to 4, 1 Gm. of digitalis leaf was given by mouth. On November 4 the atrial rate was 158 per minute. There was 1:1 A-V conduction, with a P-P interval of 0.38 second. The Q-Q interval, however, alternated between 0.37 and 0.39 second, with QRS alternation and a P-R interval alternating between 0.12 and 0.14 second (Fig. 7, *B*). On November 2, 5 grains of quinidine were given by mouth every hour for seven doses without terminating this attack. On November 5 the mechanism had changed to atrial fibrillation (Fig. 7, *C*), and this persisted until November 24. The atrial tachycardia recurred at the rate of 160 per minute on November 9; magnesium sulphate, by vein, injected for the purpose of measuring the circulation time, stopped the tachycardia immediately.

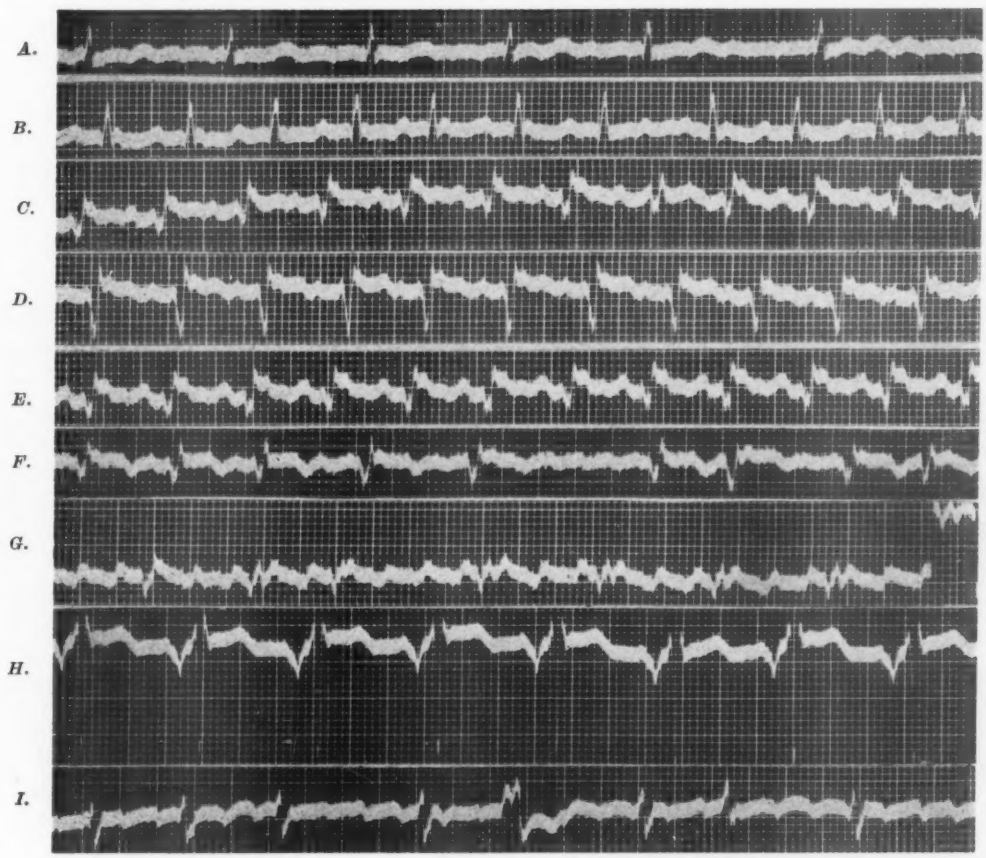

Fig. 8.—Case 8. *A*, Lead II; atrial fibrillation on Feb. 2, 1943. *B*, *C*, and *D*, Leads I, II, III, taken on February 19; showing atrial tachycardia with 2:1 A-V block. *E*, Lead II, February 20, 10:00 A.M.; tachycardia. *F*, Lead II, February 20, noon; fibrillation or impure flutter. *G*, Lead II, February 22, 9:30 A.M.; flutter. *H*, February 22, 4:00 P.M.; esophageal lead, sinus rhythm. *I*, February 23, Lead CF_1; impure flutter or fibrillation again.

On November 11, atrial fibrillation was still present (Fig. 7, *D*), although while taking an electrocardiogram one lead showed a short paroxysm of tachycardia (Fig. 7, *E*). Digitalis was withdrawn on November 10, at which time she had had a total of 1.2 Gm. by mouth,

2 mg. of digoxin, and 9 cat units of digalen. This had caused green vision and partial blindness, as well as gastrointestinal disturbances. Two weeks after digitalis was withdrawn, the administration of only 3 grains of quinidine, on November 24, caused the cardiac mechanism to revert to normal sinus mechanism (Fig. 7, *F*). By this time the urinary abnormalities had disappeared and the blood pressure had fallen to 108/74. She was seen again in January, 1943, and at that time still had sinus rhythm.

CASE 8.—S. W., a colored man, aged 64 years, John Sealy Hospital No. 66680, entered the hospital Feb. 19, 1943, because of a syncopal attack which was followed by epigastric pain which radiated into the left shoulder. He had been under observation in the Out-patient Clinic and hospital since 1937 because of hypertensive heart disease. In January, 1943, he had been hospitalized for ten days after an attack of cerebral angiospasm. On Feb. 2, 1943, he visited the clinic, and the electrocardiogram taken at that time showed atrial fibrillation (Fig. 8, *A*). At the time of admission, on February 19, clinical signs suggested that fibrillation was still present. By the time a tracing was recorded, however, the ventricular rate was regular, and the cardiac mechanism was found to have changed to paroxysmal atrial tachycardia with A-V block, usually 2:1 (Fig. 8, *B*, *C*, and *D*). On February 20 the same mechanism was present at 10:00 A.M. (Fig. 8, *E*). At 11:00 A.M. the heart had again become irregular, and the tracing taken at noon showed atrial fibrillation or impure flutter (Fig. 8, *F*). On February 22, the tracing revealed atrial flutter with variable A-V block (Fig. 8, *G*). He was given 1.4 Gm. of quinidine, which produced sinus rhythm within seven hours (Fig. 8, *H*). Flutter reappeared on the morning of February 23, and 1.6 Gm. of quinidine were given; that afternoon atrial fibrillation or impure flutter was recorded (Fig. 8, *I*). On February 24, sinus rhythm had appeared, and this mechanism persisted until his discharge in April. He died suddenly May 11, 1943, during a convulsive seizure.

CASE 9.—A. H., a colored woman, aged 46 years, John Sealy Hospital No. 19506, was admitted to the hospital Jan. 15, 1942. She had congestive heart failure. The blood pressure, which had been known to be elevated since 1926, was 250/160 on admission. During the preceding four months, she had been on irregular doses of digitalis, but during the three weeks before admission there had been progressive edema and dyspnea. The heart was markedly enlarged, and severe failure was present. Ophthalmoscopic examination showed papilledema; nitrogen retention was present. The serum albumin was 3.44 per cent, and the globulin, 6.2 per cent. There was a rectal stricture caused by lymphopathia venereum.

The electrocardiogram on the day of admission showed atrial fibrillation (Fig. 9, *A*). The next day digitalis medication was begun in a dosage of 0.3 Gm. daily, and this was continued until her death. On January 16, the pulse rate was 134 per minute, and regular. On January 18, the heart was again irregular, and a tracing taken at this time showed paroxysmal atrial tachycardia with variable A-V block (Fig. 9, *B*). On January 20, 21, and 23, there was sinus rhythm at a rate of 133 to 136 per minute, recorded electrocardiographically (Fig. 9, *C*). On January 25 there were numerous paroxysms of tachycardia, during which there was again partial A-V block. Quinidine was given without improvement (Fig. 9, *D*).

Nitrogen retention had been progressive. Pericarditis was noted first on January 20. Death occurred from uremia on Jan. 26, 1942.

CASE 10.—C. O., a white man, aged 73 years, John Sealy Hospital No. 44749, was admitted April 17, 1942, with a right-sided hemiplegia; he was semicomatose and had Cheyne-Stokes respiration. Hypertension was present (180/104), the arteries were sclerotic, and the heart was large; congestive failure was present in moderate grade. On April 17 and 18 he was given 1 mg. of digoxin intravenously; on April 19 and 20 he received 0.5 mg.; aminophyllin, in doses of 0.5 Gm., was injected April 18, 19, and 20. Electrocardiograms taken April 18, and again on April 21, showed paroxysmal atrial tachycardia with 2:1 A-V

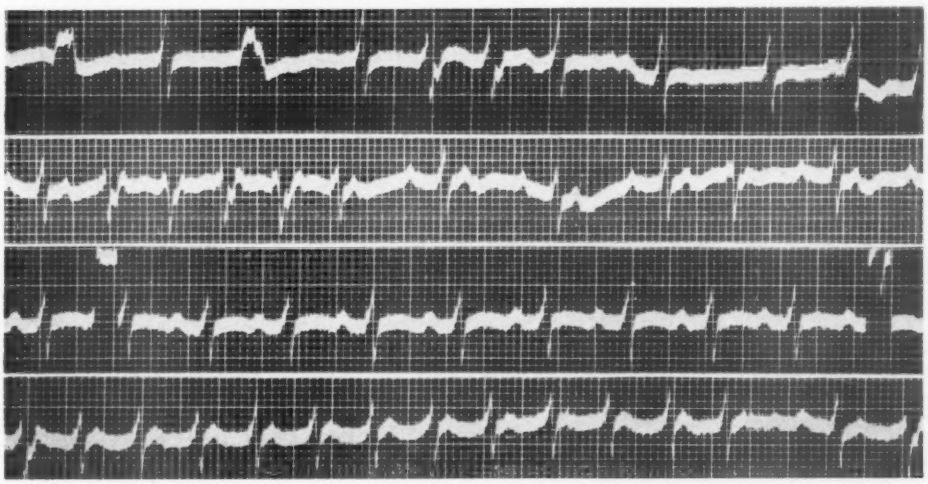

Fig. 9.—Case 9. *A*, Lead II, Jan. 15, 1942; atrial fibrillation. *B*, Lead II, January 18; atrial tachycardia with variable A-V block. *C*, Lead II; sinus rhythm on January 20. *D*, Lead II; recurrence of paroxysmal tachycardia on January 25.

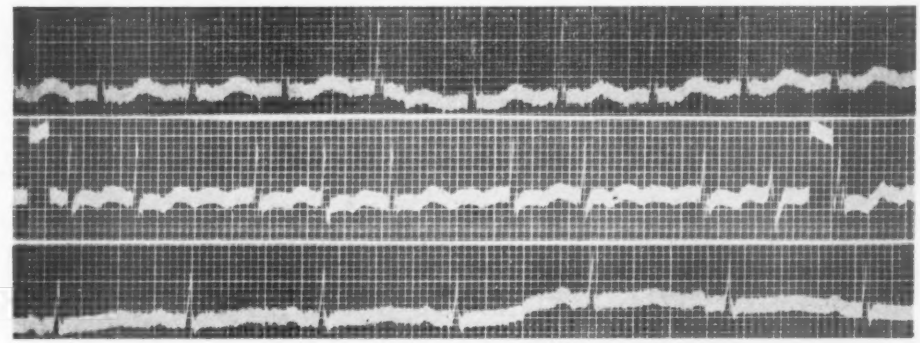

Fig. 10.—Case 10. *A*, Lead II, April 18, 1942; paroxysmal atrial tachycardia with 2:1 A-V block. *B*, Lead II, May 2; atrial flutter. *C*, Lead II; sinus rhythm on May 4.

block (Fig. 10, *A*). On April 24, frequent short paroxysms were noted clinically, although the electrocardiogram showed sinus rhythm with supraventricular premature beats. On May 2, the rhythm was irregular; the electrocardiogram revealed atrial flutter, with variable A-V block (Fig. 10, *B*); 0.6 Gm. of quinidine was given on May 3, which converted the mechanism to sinus rhythm (Fig. 10, *C*).

CASE 11.—J. S., a white man, aged 62 years, John Sealy Hospital No. 75448, was admitted July 20, 1942. He had Parkinson's disease, as well as hypertensive and arteriosclerotic heart disease, and had been hospitalized because of congestive heart failure in April, 1942. He stopped taking his maintenance dosage of digitalis about June 1, and resumed his occupation. Within two weeks, symptoms of failure reappeared, and these became progressively worse. At the time of admission the blood pressure was 160/120; the heart and liver were enlarged; edema of the lower extremities was marked. The venous pressure was 27 cm. of saline.

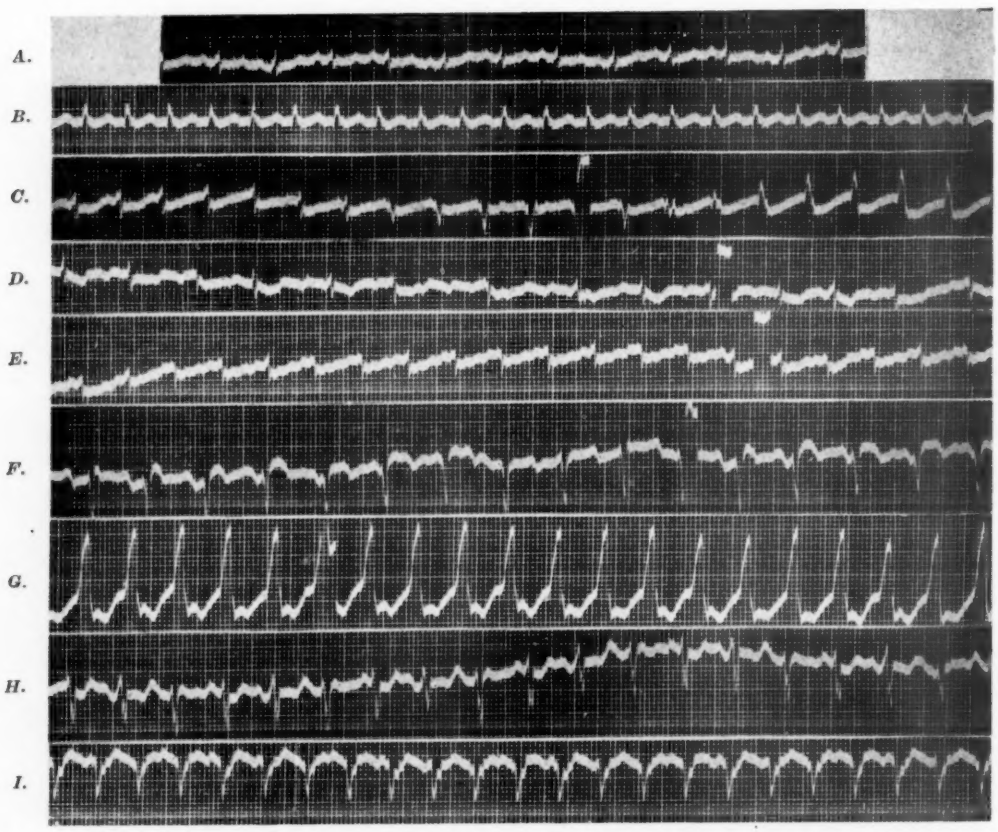

Fig. 11.—Case 11. *A*, Lead II, July 21, 1942; sinus rhythm. *B*, Lead II, July 23, 2:00 A.M.; paroxysmal atrial tachycardia. *C*, July 23, 1:17 P.M., Lead II; tachycardia with slower rate and much aberration of the QRS complexes. *D*, 2:50 P.M., Lead II; flutter with variable A-V block. *E*, 3:10 P.M., Lead II; flutter with 2:1 block. *F*, Lead CF_1, July 24; auricular tachycardia, with A-V block and independent ventricular rhythm. *G*, Lead II, July 25, morning; probably atrial tachycardia with QRS aberration. *H*, Lead II, July 25, afternoon; atrial tachycardia with variable rate, and probably complete A-V block. *I*, Lead II, July 27; showing the reappearance of flutter.

The cardiac rhythm was regular (Fig. 11, *A*), with atrial premature beats which were noted, clinically, to occur in frequent short runs. Pulmonary infarction with hemoptysis developed on July 21. Digitalis in a dose of 1.2 Gm. was given from July 20 to 23.

At 1:00 A.M. on July 23 he was found to have what was probably paroxysmal atrial tachycardia, with a rate of 170 per minute (Fig. 11,

B); carotid sinus pressure and the injection of 1 mg. of prostigmine were without effect; 5 mg. of neosynephrin were likewise ineffectual. At 1:00 P.M. of July 23, the rate had slowed to 150 per minute; there was marked, transient variation in the QRS complexes, with varying degrees of aberration (Fig. 11, *C*). At 2:50 P.M. atrial flutter was present with variable block (Fig. 11, *D*); 10 mg. of neosynephrin were given, and at 3:10 P.M. there was a regular 2:1 A-V block (Fig. 11, *E*); 2.1 Gm. of quinidine were given during the night, and the next day, July 24, the atrial rate had dropped to 240 per minute (Fig. 11, *F*); there was complete A-V dissociation, with an idioventricular rhythm at a rate of 122 per minute. On the morning of the 25th the heart rate was 150 per minute (Fig. 11, *G*); the mechanism may be ventricular tachycardia, but in view of the marked aberration of the QRS complex which had previously been present, we regard these as being of supraventricular origin, with defective bundle branch conduction; the P waves appear to be in the S-T segment. Two and four-tenths grams of quinidine were given in divided doses, and by late afternoon of July 25 the mechanism was again changed. The atrial rate varied from 210 to 250 per minute; the ventricular rate was 140, with probably complete A-V block (Fig. 11, *H*). This tracing is interpreted as representing a return to the atrial tachycardia that was previously present.

Prostigmine and quinidine were given repeatedly on July 26, but the heart rate remained between 140 and 150; 0.4 Gm. of digitalis produced no change in rate. The tracing taken on July 27 shows a ventricular rate of 167 per minute; atrial flutter is present at a rate of 300 per minute (Fig. 11, *I*). Death occurred July 28.

Necropsy showed a heart weighing 470 grams. Coronary arteriosclerosis was present, and, although no coronary thrombosis was found, there was a myocardial infarct at the tip of the left ventricle. Mural thrombi were present in the left ventricle adjacent to the myocardial infarct, and also in the right atrium. There were embolic infarcts of the lungs, and of the kidneys, spleen, and brain.

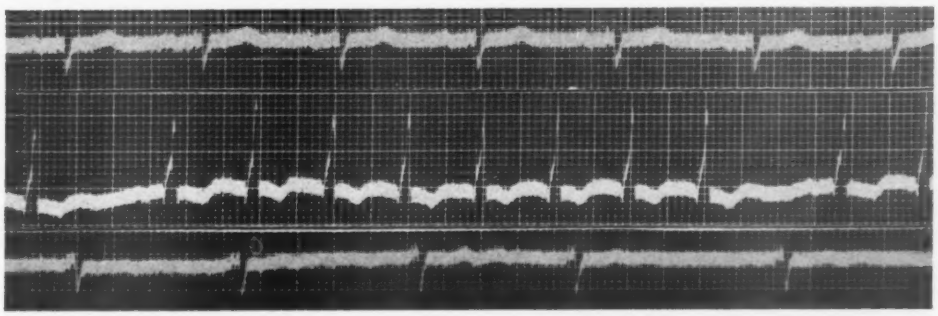

Fig. 12.—Case 12. *A*, Lead II, Aug. 28, 1942; sinus rhythm. *B*, Lead IVF, August 30; showing a short paroxysm of tachycardia. *C*, Lead II, September 1; atrial fibrillation.

CASE 12.—M. G., a white man, aged 46 years, John Sealy Hospital No. 77524, entered the hospital Aug. 27, 1942, with congestive heart failure of four months' duration due to arteriosclerotic heart disease. Digitalis medication was as follows: August 29, 0.6 Gm., August 30, 31, and September 1, 0.4 Gm.; beginning September 2, 0.1 Gm. daily. On August 28, the electrocardiogram showed sinus rhythm at a rate of

79 per minute (Fig. 12, *A*); the situation was the same on August 29. The tracing taken August 30 (Fig. 12, *B*) showed frequent short paroxysms of supraventricular tachycardia, at a rate of 150 per minute,

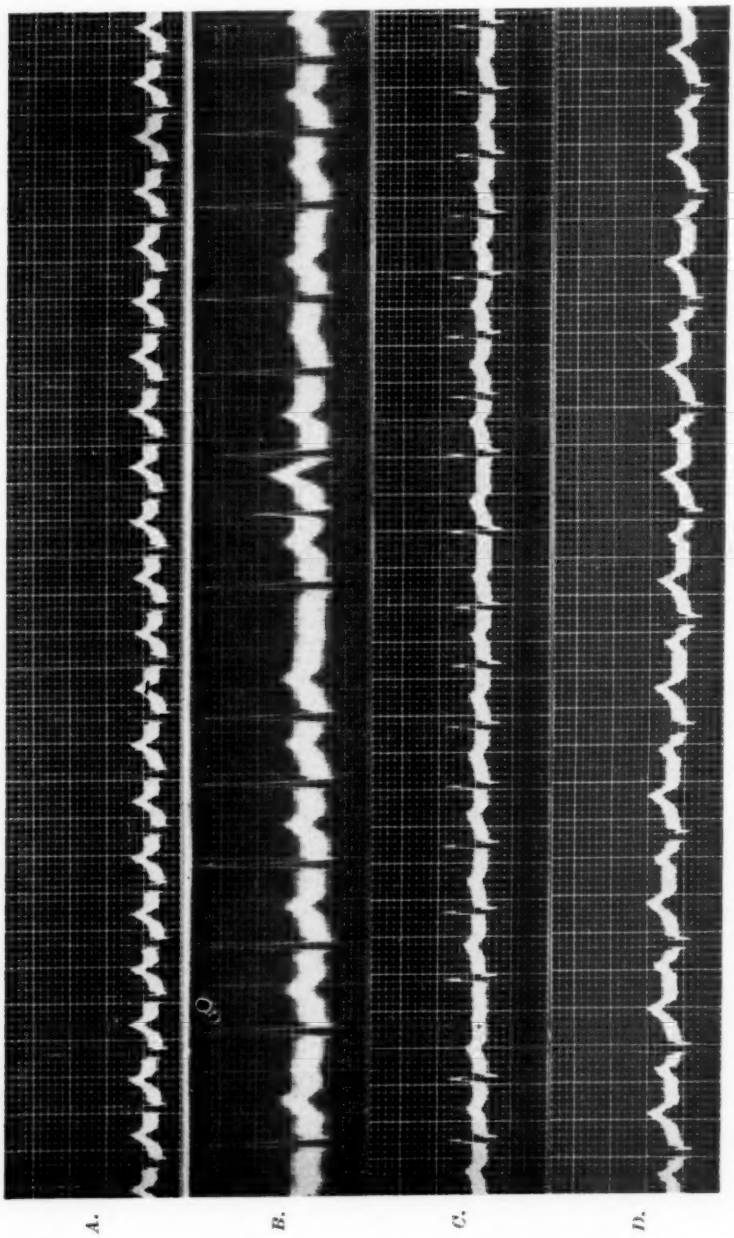

Fig. 13.—Case 13. *A*, Lead II, Nov. 17, 1942; paroxysmal atrial tachycardia. *B*, Lead II, June 10, 1943; atrial fibrillation, immediately after the spontaneous termination of a typical paroxysm of tachycardia. *C*, Lead II, June 10; three hours after *B*. *D*, Lead II, June 10; sinus rhythm; one hour after 3 grains of quinidine.

in some of which there was partial A-V block. The curves taken the next day, August 31, disclosed that the mechanism had changed to atrial fibrillation (Fig. 12, *C*). The fibrillation persisted until October 21; on this day sinus rhythm reappeared, and persisted.

CASE 13.—J. H., a white man, aged 48 years, John Sealy Hospital O.C. No. 112683, attended the clinic for antisyphilitic treatment. He had had a penile chancre, in 1916, which had been treated locally with calomel. A recent blood Wassermann reaction had been found to be positive. When he was examined on Nov. 11, 1943, his heart was considered normal; the blood pressure was 136/86. However, he gave a history of having had paroxysms of tachycardia for the preceding twenty-five years. These varied in frequency as well as duration; they lasted from ten minutes to several hours, and often stopped spontaneously. On other occasions the paroxysms could be terminated by bending, straining, or gagging. While visiting the clinic on November 17 he had a typical paroxysm. Electrocardiograms were obtained, and showed paroxysmal supraventricular tachycardia at a rate of 231 per minute (Fig. 13, *A*). Carotid sinus pressure was without effect. Prostigmin in a dose of 1 mg. was injected, and fourteen minutes later the paroxysm was terminated. After the paroxysm, the electrocardiogram showed no abnormalities.

Between November, 1942, and June, 1943, he was given twenty injections of bismuth and four injections of an arsenical. On June 10, he was again seen in the clinic in a typical attack of paroxysmal tachycardia. The rate was 250 per minute, and the rhythm was regular. Before the electrodes could be applied the patient volunteered the information that the attack had ceased spontaneously. When his pulse was felt it was found to be much slower, but irregular. The electrocardiogram taken at this time, 11:30 A.M., showed atrial fibrillation, with a ventricular rate of about 160 per minute (Fig. 13, *B*). He was observed for three hours in anticipation of spontaneous reversion of the fibrillation; at 2:30 P.M. fibrillation was still present (Fig. 13, *C*). A test dose of 3 grains of quinidine was given; an hour later normal sinus rhythm was present (Fig. 13, *D*).

CASE 14.—P. J., a colored man, aged 76, John Sealy Hospital No. 78053, was admitted March 1, 1943, because of congestive heart failure which had gradually become manifest during the preceding two years. For the preceding eighteen days dyspnea and edema had become severe. The blood pressure was 200/90; the heart was enlarged, and there were the usual evidences of heart failure of moderate grade. The venous pressure was 21.5 cm. of saline. The electrocardiogram on March 2 showed sinus rhythm with frequent ventricular premature beats. Digitalis was begun on March 2, and, by March 5, 1.2 Gm. had been given; after this day, 0.1 Gm. was given daily. By March 5 atrial fibrillation was present, and was recorded on March 9 and again on March 16 (Fig. 14, *A*). Quinidine was administered in a dose of 0.6 Gm. daily from March 3 to 17; on March 18, 2 Gm. were given; from March 19 to 26 the 0.6 Gm. dosage was resumed.

On March 20 the electrocardiogram showed paroxysmal atrial tachycardia with an atrial rate of 166 to 171 per minute, and with 2:1 and 3:2 A-V block. The tachycardia was still present on March 22, with 2:1 A-V block at an atrial rate of 214 per minute; an hour later the atrial rate had dropped to 162, and 1:1 conduction occurred; at this time 1 mg. of prostigmin was given, and, although the atrial rate was only 171 to 179 per minute, A-V block appeared, varying from 3:2 to 2:1 (Fig. 14, *B*).

The tracing taken March 23 is of particular interest, and all of Lead II is reproduced (Fig. 14, *C* and *D*). The atrial rate varies from 260

Fig. 14.—Case 14. A, Lead II, March 9, 1943; atrial fibrillation. *B*, Lead II, March 22; showing tachycardia with A-V block. *C* and *D*, Continuous strips of Lead II, March 23; showing variation in atrial rate, and possibly cyclic variation between tachycardia and flutter. *E*, Lead II, March 25; tachycardia with A-V block. *F*, Lead II; sinus rhythm on March 29. *G*, Lead II; atrial fibrillation again on May 11, 1943.

to 316; at the slower rates the tracing seems definitely to be atrial tachycardia; at the more rapid rates the presence of flutter seems equally certain. Although it is difficult to be positive, the curve is very suggestive of cyclic variation between atrial tachycardia and atrial flutter. The tracings taken on March 25 and 26 showed tachycardia with partial A-V block, usually 2:1 (Fig. 14, *E*). On March 29th, sinus rhythm was present (Fig. 14, *F*). The patient was discharged April 4, 1943, on a maintenance dose of digitalis.

He returned to the Out-patient Clinic on April 27 with extreme cardiac failure. The electrocardiogram showed a return of atrial fibrillation. Diuresis was obtained with ammonium chloride and mercupurin, with clinical improvement. On May 4, the mechanism had changed to sinus rhythm. On May 11, however, the digitalis was discontinued because of nausea and vomiting; on this day the tracing showed atrial fibrillation (Fig. 14, *G*). He was not seen again until June 3, at which time the severity of his cardiac failure necessitated hospitalization. The last electrocardiogram showed sinus rhythm. He died June 4, 1943.

COMMENT

The intimate relationship of atrial flutter and fibrillation is well recognized. The older theories of the mechanism of these disturbances have been reviewed by Garrey,[11] who also summarized the available evidence in favor of the theory of a circus mechanism. This latter concept has found wide acceptance, although the Viennese group of cardiologists prefer the concept of one or more rapidly discharging ectopic or parasystolic foci operating to produce either flutter or fibrillation. The circus mechanism may be modified by assuming a single initial premature stimulus, followed by multiple re-entries. Lack of conclusive evidence for any one of these possible mechanisms does not vitiate the fact that the same fundamental mechanism appears to be operative for both flutter and fibrillation. This is amply substantiated by both laboratory and clinical experience.

Paroxysmal atrial tachycardia, however, has been generally thought to be the result of a totally different mechanism. The most widely held view is that paroxysms result from the rapid discharge of impulses from an ectopic pacemaker. Multiple re-entries from a single impulse, and the removal of blocking from a parasystolic focus have also been proposed. The views concerning the possible mechanism of paroxysmal ventricular tachycardia, many of which are equally applicable to the atrial variety, have recently been reviewed by Cooke and White.[12]

Suggestions have not been entirely lacking that paroxysmal tachycardia is due to a mechanism similar to that responsible for flutter and fibrillation. Iliescu and Sebastiani[13] noted the similarity between the action of quinidine in cases of paroxysmal tachycardia and its effect in flutter and fibrillation. They stated that quinidine produces a marked slowing of the atrial rate in fibrillation, less in flutter, and still less in tachycardia, and considered that the variation in its action was quantitative rather than qualitative, and might depend in part on the original

atrial rate. The preliminary slowing of the atrial rate, and subsequent abrupt transition to normal mechanism, suggested to them the possibility that paroxysmal tachycardia, as well as flutter and fibrillation, might be the result of a circus mechanism.

Ashman and Hull[14] have given as their view the concept that paroxysmal tachycardia is due in many instances to a circus mechanism which passes through the S-A or A-V node. If this were so, the susceptibility of the tachycardia to termination by reflex vagal stimulation would result from vagal influence on the nodal portion of the circuit. Presumably, also, the isoelectric period of the electrocardiogram, the chief criterion for differentiation from flutter, would coincide with the relatively slow passage of the impulse through the nodal tissue.

More recently, Barker, et al.,[15] have discussed the similarity, as well as the differences, between tachycardia and flutter-fibrillation. They believe that paroxysmal tachycardia, particularly in those cases in which there is either A-V block or alternation of the P-P cycle length, may best be explained by assuming that a circus mechanism passes through the S-A or A-V node. The experimental reproduction by Butterworth and Poindexter,[16] of the Wolff-Parkinson-White syndrome of short P-R interval with prolonged QRS complex indicates strongly that there is a circus movement during the paroxysms of tachycardia which are typically associated with this syndrome. Presumably the circus moves down the A-V conduction tissue, and re-enters the atrium through the bundle of Kent.

We do not believe that our own data can be used as evidence in favor of any one of the hypotheses for the fundamental mechanism of these disturbances. However, the fact seems clearly established that some cases of paroxysmal atrial tachycardia are closely related to flutter and/or fibrillation.

This is particularly true of those cases of tachycardia in which partial A-V block has been found to exist at some time, thus confirming the same observation by Barker and his associates.

Whether this is a coincidence, or actually indicates a circus mechanism in these cases of paroxysmal tachycardia, must be ascertained by more definitive means. As an initial effort in this direction, we have recently calculated the electrical axes of the P waves at $\frac{1}{100}$ second intervals, using simultaneous chest leads placed in three planes, as was done by Lewis, et al.[17] Judging from our experience to date,[18] we believe that the method is capable of showing the existence of a circus movement, if one be present. At least, our results in flutter and fibrillation seem to agree in general with those obtained by Lewis. However, in four cases of paroxysmal atrial tachycardia with A-V block, the curves representing the consecutive electrical axes of the atrium fail to suggest a circus movement; they rather resemble the curves obtained from individuals with normal sinus rhythm, differing only in their direction. These data will be reported soon in detail.

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MEASUREMENTS OF ARTERIAL BLOOD PRESSURE IN THE ARM AND LEG: COMPARISON OF SPHYGMOMANOMETRIC AND DIRECT INTRA-ARTERIAL PRESSURES, WITH SPECIAL ATTENTION TO THEIR RELATIONSHIP IN AORTIC REGURGITATION

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THE commonly used clinical methods of measuring arterial blood pressure are based on the concepts proposed originally by Riva-Rocci,[1] von Recklinghausen,[2] and Korotkow.[3] Examination of the original articles of these writers reveals that their technique is founded on indirect evidence. Many attempts have been made to study the accuracy of clinical methods of measuring blood pressure, but, prior to 1931, these could be based only on theoretical deductions. In that year, Wolf and von Bonsdorff[4] introduced a method for accurate optical recording of human blood pressure by arterial puncture, thereby permitting direct observation of the accuracy of bloodless methods.

The various factors influencing the measurement of blood pressure in the brachial artery by the cuff method have been studied in adults[5] and children[6] by Hamilton and his co-workers, and more recently in adults by Ragan and Bordley.[7] These workers have found that the standard arm cuff (12 to 13 cm. wide) does not permit accurate measurement of blood pressure in all subjects, and that no definite method of prediction permitted choice of a cuff of proper width, either for children or some adults.

Although Hales,[8] in 1733, employed the femoral artery of a horse in the first recorded direct measurement of blood pressure, and although many investigators have used it in animal experiments since, there have been relatively few studies of femoral blood pressure in man by direct methods. Thus, the accuracy of clinical methods of measurement of femoral blood pressure cannot be regarded as established. In 1939, The American Heart Association and the Cardiac Society of Great Britain and Ireland[9] recommended a cuff 15 cm. wide for use on the thigh. The exact reasons for this choice were not given, although Recklinghausen[2] had stated that a cuff of 15 cm. was wide enough for any extremity. However, there is no experimental verification that this or any other cuff is adequate for accurate measurements of femoral blood pressure.

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There is little agreement regarding the relation of femoral and brachial blood pressure. The usual clinical concept is that the femoral systolic blood pressure is approximately equal to the brachial systolic blood pressure in normal persons, but that in aortic insufficiency and under certain other conditions it is considerably higher. Hamilton and his co-workers[5] demonstrated by arterial puncture that the femoral systolic blood pressure is higher than the brachial in normal persons, although he did not specify the number of subjects studied. The femoral diastolic pressure was found to be the same or slightly lower than the brachial diastolic pressure. Wolf and Bonsdorff,[4] on the other hand, compared the femoral pressure, as ascertained by arterial puncture, with the arm pressure, as ascertained by the usual Korotkow method, in six subjects without valvular disease or hypertension. In an analysis of this material, they concluded that "there could be no great difference between the femoral and brachial pressure" in man.

In the present study, blood pressure was measured in the brachial and femoral arteries of subjects with normal pressures, hypertension, and aortic insufficiency by cuff methods and by use of the optical manometer. We desired, first, to test the accuracy of the 15 cm. cuff for measurements of blood pressure in the thigh; second, to test the validity of the impression that aortic regurgitation produces a disproportionate elevation of the systolic pressure in the leg (thigh); and, third, to compare the respective femoral pressures with the concomitant pressures in the arm.

METHOD

Direct measurements of femoral and brachial blood pressure were made on cooperative, convalescent subjects from the hospital wards. Repeated measurements with the cuff were made on the extremity in question until the blood pressure range was stable, before, during, and after the optical manometric measurements. Systolic levels were read at the first appearance of sound, and diastolic levels at the point at which sounds become dull and muffled (between third and fourth phases). For the arm the standard 13 cm. cuff was used; for the leg we employed both the standard arm cuff (called the "narrow leg cuff" in this report), as well as a specially made cuff, 15½ cm. wide, tapered 4 cm. at this lower edge for better fit. The cloth covering of the wider leg cuff was 2 cm. wider and 2 cm. longer than the rubber bag, and the cuff wrapper measured 130 cm. in length. this varies but slightly from the specifications proposed for the leg cuff by The American Heart Association and the Cardiac Society of Great Britain and Ireland.

Direct intra-arterial measurements were made with the Hamilton manometer as modified by Gregg, et al.[10] The membranes used were usually rubber* (cardiovascular sheeting, 0.028 inch thick), although glass spoons† were used in a few experiments. Neither possessed

*Generously supplied by Dr. A. Szegvari of the American Anode, Inc.
†Kindly furnished by Dr. H. F. Helmholz, Rochester, Minnesota.

greater advantage in our hands. The frequency of these membranes, as tested before each experiment, was at least 80 double vibrations per second; Frank[11] considered 40 double vibrations per second adequate for recording most of the peripheral pulse wave characteristics.

The light source was a 500 watt Mazda projection light, placed 12 cm. behind an adjustable vertical slit, and directed upon a small plane mirror mounted on the membrane which the arterial pulsations moved. The image was focused by a convex lens of low power upon the revolving paper film of the Cambridge research model electrocardiograph, 140 cm. distant. The apparatus was adjusted so that the level of the artery to be punctured was on the same plane as the fluid levels in two pressure reservoirs, one of which contained 5 per cent sodium citrate solution, and the other, water; readings were thus corrected automatically for the level of the puncture site.

For the procedure itself a cooperative patient was placed supine on a flat table and the extremity to be studied was placed comfortably in a natural position. The puncture site in the brachial artery was the antecubital fossa, just above the anterior crease of the elbow, and, in the femoral artery, the femoral triangle just below the inguinal ligament. Before puncture, these areas were cleansed with iodine and alcohol, and the skin and subcutaneous tissue, down to the artery, were infiltrated with 1 per cent procaine solution. The only pain which the subjects noted was felt as a brief twinge at the moment of arterial puncture.

After blood pressures were taken with the proper cuff and found stable, the artery was punctured, using a sterile, short bevelled, No. 21 needle, 1⅝ inches in length over all. The needle was inserted into the artery with the bevel close to the perpendicular. After graphic registration of the blood pressure, the cuff measurements were repeated; in the case of the arm, checks were made at some point while the manometer was recording.

Graphic records were taken at two speeds; pressure and time measurements were made from the higher speeds for greater accuracy. The membrane was then calibrated in millimeters of mercury with the needle in place, using a mercury-filled U tube and the pressure bottle; calibrations were made every 50 mm. Hg up to a pressure of 250 mm. Hg. After calibration, another graphic record was usually made, unless the citrate in the tubing was inadequate to prevent clotting during this period. It is worth noting that none of the citrate entered the circulation or surrounding tissue. After removing the needle, firm compression was applied to the artery for five minutes.

If, as infrequently happened, the artery could not be readily entered, the experiment was immediately abandoned, rather than risk injury to the vessel or excitement to the patient.

Simultaneous registration of the femoral and brachial blood pressures was not done, but they were measured in quick succession (about 5 to 8 minutes apart) without apparent change in the patient's mental or physical state. In about half the experiments, the arm pressures were measured first, and in the remaining group, the leg measurements were made first.

RESULTS

Measurements were done on nine patients with normal blood pressure, nine patients with hypertensive cardiovascular disease, and ten

patients with aortic insufficiency. The results are listed in Table I. In all instances in which there was a range in cuff pressures* after the initial fluctuations had disappeared, a range is listed. All subjects, except one, showed some variation (mainly respiratory) in the height of the manometric recordings; this was pronounced in some and slight in others, but always more marked in the systolic level. For purposes of comparison, average cuff pressures were compared with average manometric pressure. Differences of less than 10 mm. of mercury were considered within the limits of error of the graphic method, because of experimental conditions.

Reference to Table II will show the differences between direct pressures in the brachial and femoral arteries and the percentage error from use of the respective cuffs on the arm and thigh. In this table and the following discussion the values obtained by direct methods are considered to represent the true intra-arterial pressure; and the results from use of the cuff are taken to be the values which may deviate one way or the other from the true direct pressure. Cuff and direct readings on the normal subjects show a fair agreement in the brachial systolic pressures, except in two subjects. The brachial diastolic cuff pressures were usually disproportionately higher; others[5, 7] have noted the character and direction of this disagreement. In the subjects with normal blood pressures, both the narrow and the wide leg cuffs gave results which were usually too high for the systolic, and always considerably higher for the diastolic pressures, when these values were compared with manometric readings.

Cuff measurements and direct brachial recordings on the subjects with hypertension showed a fair agreement of the systolic pressures in all but two patients, in both of whom the cuff pressures were too low (minus 27 and minus 20 mm. mercury). The brachial diastolic cuff pressures were too high in five of the nine subjects in this group. Again, the narrow cuff gave readings of the femoral blood pressures which were consistently too high for both the systolic and diastolic levels. The wide cuff permitted a somewhat better estimation of the femoral systolic pressure in those patients on whom it was used; the diastolic readings with the wide cuff were uniformly too high, averaging 14 mm. above graphic pressure.

Cuff and graphic arm readings on the patients with aortic insufficiency showed a fair agreement in the systolic level. The brachial diastolic pressures agreed in only one subject (Case 2). In two patients with aortic regurgitation (Cases 6 and 9), whose sharp arterial sounds were heard down to zero pressure in both arm and leg, the true brachial diastolic pressure was found to be 41 mm. and 35 mm. Hg, respectively. Femoral wide cuff pressures were consistently too

*In the following discussion the words "direct," "manometric," and "graphic" refer to arterial blood pressures obtained by use of the optical manometer; "cuff" pressure refers to that obtained by use of the arm or leg cuffs, mercury manometer, and the auscultatory technique.

TABLE I

CASE NUMBER	NAME	DIAGNOSIS	BRACHIAL				FEMORAL				
			CUFF PRESSURE	GRAPHIC RANGE	GRAPHIC AVERAGE	PULSE RATE AVERAGE	WIDE CUFF PRESSURE	NARROW CUFF PRESSURE	GRAPHIC RANGE	GRAPHIC AVERAGE	PULSE RATE AVERAGE
			Control Group								
1	M. H.	Convalescent pneumonia	115 / 70	122-116 / 63-58	119 / 61	56	135 / 90	150 / 90	130-128 / 59-55	129 / 57	58
2	A. R.	Syphilitic cerebral thrombosis	106 / 84-78	107-105 / 67-63	106 / 65	77	120 / 78	155 / 105	105-99 / 59-55	102 / 57	77
3	J. M.	Peptic ulcer (post bleeding)	126-105 / 65-56	94-92 / 47	93 / 47	74		124 / 80	105-93 / 34	99 / 34	70
4	C. Z.	Convalescent pneumonitis	118-105 / 66-60	124-110 / 63-57	117 / 60	75	132-130 / 74-70	132 / 78	139-123 / 63-53	131 / 58	81
5	G. C.	Cholangitis	110 / 75-70	117-110 / 66-62	113 / 64	55	120 / 90	130 / 100-95	133-118 / 70	125 / 70	56
6	C. P.	Rheumatic fever	125-120 / 75-70	129-119 / 71-66	124 / 69	86	135-130 / 95-90	145-140 / 95-90	138-129 / 68-61	134 / 65	80
7	R. M.	Rheumatic fever	120 / 70	138-130 / 77-74	134 / 76	96	135-130 / 80-75	155 / 105	130-124 / 65	127 / 65	96
8	Z. M.	Convalescent pneumonia	140-135 / 85	133-130 / 90-84	132 / 87	110	155-150 / 95-90	165 / 105	159-148 / 86-77	154 / 82	112
9	W. J.	Uncontrolled diabetes	135-130 / 90-85	136-132 / 83-78	134 / 81	80	135-130 / 95	165-160 / 110-105	140-125 / 80-73	133 / 77	77
			Hypertensive Patients								
1	P. A.	Hypertensive heart disease (mild failure)	188-170 / 105-100	215-203 / 95-88	209 / 92	55	238-235 / 110-108	270 / 130	224-215 / 81-74	219 / 78	57
2	C. H.	Hypertension	210-205 / 140	214-206 / 129-119	210 / 124	86		260 / 160	222-214 / 131-123	218 / 128	92
3	W. J.	Hypertension hemiplegia	200 / 155	198 / 126	198 / 126	89		260 / 180	215-211 / 123	213 / 123	86
4	S. W.	Hypertension	195-190 / 108	215-190 / 112-107	196 / 110	96		240 / 140	220-196 / 109-89	208 / 99	96

5	M. M.	Rheumatoid arthritis	180-168 82-78	183-158 85-75	171 80	83	170-165 85		176-162 71-62	169 67	80
6	W. H.	Hypertension hemiplegia	173-165 120-115	192-185 115-108	189 112	97	195-180 135-120		200-187 114-111	193 113	90
7	M. R.	Hypertensive encephalopathy	155-146 108-98	143-131 94-87	137 91	84	185-160 120-100	245 160	143-130 91-82	137 87	79
8	J. A.	Diabetes	190-180 120-115	204-188 115-103	196 109	98	205-200 120	210-200 125	197-175 115-106	186 111	96
9	J. R.	Carcinoma of stomach	165-155 90-85	164-148 82-76	156 79	94	180 100		171-163 87-79	167 83	93
		Patients With Aortic Insufficiency									
1	M. C.	Syphilitic aortic regurgitation	140-135 35-30	135-128 21-16	132 19	82	215 30	250-235 50-35	161-149 17-7	155 12	81
2	J. B.	Syphilitic aortic regurgitation	160-145 50-45	155-124 57-33	140 45	74	240 55	295-275 60-55	183-146 43-24	165 34	79
3	G. N.	Syphilitic aortic regurgitation	145 55	148-142 40-35	145 38	90		210-205 80-70	152-142 35-30	147 33	92
4	R. G.	Syphilitic aortic regurgitation	170 85	187-178 72-66	183 69	66		200 105	207-197 81-71	202 76	75
5	A. C.	Aortic regurgitation	170-160 68-60	172-163 52-47	168 50	84		175 70	161-150 45-41	156 43	84
6	S. M.	Rheumatic aortic regurgitation	134 0	139-130 43-39	134 41	72	200 0	210 0	150-140 46-39	145 43	70
7	G. M.	Acute aortic endocarditis	112 44	104-100 41-38	102 39	77	145 55		116-107 36-34	112 35	78
8	W. G.	Syphilitic aortic regurgitation hypertension	160-155 105	149-142 93-90	146 92	85	188 118	210 125	176-159 92-81	168 87	82
9	T. G.	Syphilitic aortic regurgitation	145-136 0-0	137-124 40-30	131 35	90	230 0	240 0	200-178 37-34	189 36	89
10	M. W.	Syphilitic aortic regurgitation	165-155 60	175-170 45-41	173 43	86	195-185 50-45	215-205 55-50	160 37	160 37	84

TABLE II

CASE NO.	NAME	DIAGNOSIS	DIRECT LEG/ARM DIFFERENCE (SYSTOLIC/DIASTOLIC) (MM. HG.)	BRACHIAL ARM CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)	FEMORAL	
					WIDE (15.5 CM.) CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)	NARROW (12 CM.) CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)
		Control Group				
1	M. H.	Convalescent pneumonitis	+10 − 4	− 3 +14	+ 4 +58	+16 +58
2	A. R.	Syphilitic cerebral thrombosis	− 4 − 8	0 +24	+17 +37	+52 +84
3	J. M.	Peptic ulcer	+ 6 −13	+24 +29		+25 +135
4	C. Z.	Convalescent pneumonitis	+14 − 2	− 4 + 5	0 +26	0 +34
5	G. C.	Cholangitis	+12 + 6	− 2 +14	− 4 +22	+4 +40
6	C. P.	Rheumatic fever	+10 − 4	0 + 5	0 +30	+ 7 +43
7	R. M.	Rheumatic fever	− 7 − 9	−10 − 8	+ 4 +20	+22 +62
8	Z. M.	Convalescent pneumonitis	+22 − 5	+ 4 − 2	0 +11	+ 7 +28
9	W. J.	Uncontrolled diabetes	− 1 − 4	0 + 7	0 +19	+23 +40
AVERAGE CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)				+ 1.0 + 9.7	+ 2.6 +28	+17 +58
		Hypertensive Patients				
1	P. A.	Hypertensive heart disease (mild failure)	+10 −14	−17 +12	+ 9 +40	+23 +66
2	C. H.	Hypertension	+ 8 + 4	0 +13		+19 +25
3	W. J.	Hypertension hemiplegia	+15 − 3	0 +23		+22 +46
4	S. W.	Hypertension	+12 −11	0 0		+15 +41
5	M. M.	Rheumatoid arthritis	− 2 −13	0 0	0 +27	
6	W. H.	Hypertension hemiplegia	+ 4 + 1	−11 + 5	− 3 +13	
7	M. R.	Hypertensive encephalopathy	0 − 4	+10 +13	+26 +26	+78 +84
8	J. A.	Diabetes	−10 + 2	− 6 + 8	+ 9 + 8	+10 +13
9	J. R.	Carcinoma of stomach	+11 + 4	+ 3 +11	+ 8 +21	
AVERAGE CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)				− 2 + 9	+ 8 +23	+28 +46

TABLE II—CONT'D

CASE NO.	NAME	DIAGNOSIS	DIRECT LEG/ARM DIFFERENCE (SYSTOLIC/DIASTOLIC) (MM. HG.)	BRACHIAL ARM CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)	FEMORAL	
					WIDE (15.5 CM.) CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)	NARROW (12 CM.) CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)
Patients With Aortic Insufficiency						
1	M. C.	Syphilitic aortic regurgitation	+23 − 7	+ 5 +74	+39 +150	+54 +258
2	J. B.	Syphilitic aortic regurgitation	+25 −11	+ 9 + 7	+45 +62	+73 +71
3	G. N.	Syphilitic aortic regurgitation	+ 2 − 5	0 +45		+41 +127
4	R. G.	Syphilitic aortic regurgitation	+19 + 7	− 7 +23		0 +38
5	A. C.	Syphilitic aortic regurgitation	−12 − 7	− 2 +28		+12 +63
6	S. M.	Rheumatic aortic regurgitation	+11 + 2	0 -	+38	+45 -
7	G. M.	Acute aortic endocarditis	+10 − 3	+10 +13	+29 +57	
8	W. G.	Syphilitic aortic regurgitation hypertension	+22 − 5	+ 8 +14	+12 +36	+25 +44
9	T. G.	Syphilitic aortic regurgitation	+58 + 1	+ 8 -	+22 -	+27 -
10	M. W.	Syphilitic aortic regurgitation	−13 − 6	− 8 +40	+19 +30	+31 +43
AVERAGE CUFF ERROR (SYSTOLIC/DIASTOLIC) (%)				+ 2.3 +24	+29 +67	+34 +92

high, especially for the systolic, but also for the diastolic, level. These differences were greatly exaggerated by the use of the narrow cuff. The femoral diastolic pressures in the same two subjects whose cuff readings on arm and leg were zero were 43 and 36 mm. Hg, respectively. The femoral pressure with the wide or narrow cuff approached accuracy in only one systolic reading and in no diastolic readings in the patients with aortic regurgitation.

Comparison of the graphic blood pressures in the arm and leg showed a noteworthy elevation of systolic pressure (+10 mm. Hg, or more) in the femoral artery in five of the nine control subjects. In the hypertensive group a noteworthy elevation of the femoral over the brachial systolic graphic pressures was noted in four of the nine patients; one subject presented a lower systolic pressure (−10 mm.) in the leg than in the arm. In aortic regurgitation the graphic systolic pressure was significantly higher in the leg in seven of the ten cases studied; in two patients the graphic systolic pressure was lower in the leg than in the arm. The graphic diastolic pressures in the arm and leg showed less consistent differences. However, the femoral

diastolic pressure was definitely lower in the leg than in the arm in one control subject (–13 mm. Hg), in three hypertensive subjects (–14, –11, and –13 mm. Hg), and in one patient with aortic regurgitation (–11 mm. Hg). In the other cases these figures generally agreed except for slight and relatively unimportant differences.

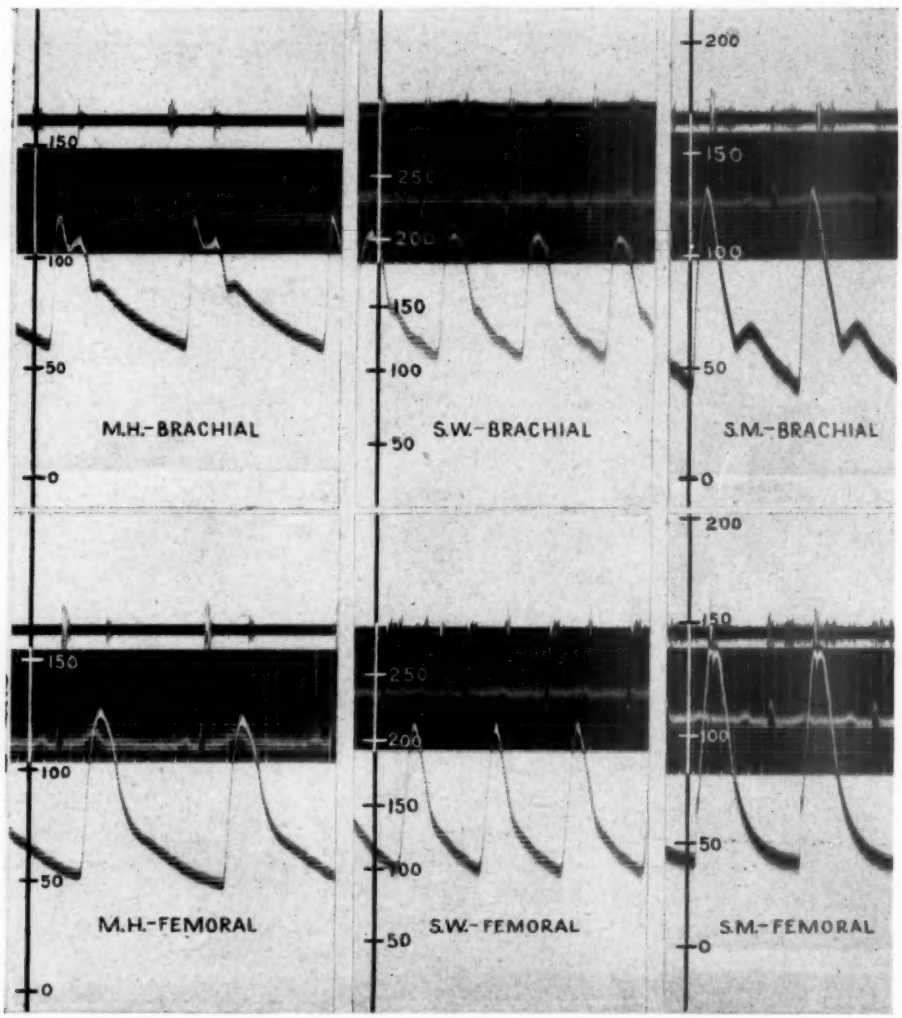

Fig. 1.—Pulse curves of subjects with normal pressure, hypertension, and aortic regurgitation, reading from left to right. The apices of the curves have been retouched for greater clarity in reproduction. The electrocardiogram and heart sounds are also shown.

The form of the brachial and femoral curves is shown in Fig. 1. In general, the contour of these curves is similar to those described by Wiggers,[12] Wolf and von Bonsdorff,[4] Hamilton, et al.,[5] and others. When the pulse curves of all patients were plotted on a common

time versus pressure graph, seven patients with aortic regurgitation showed a steeper upstroke than the control or hypertensive group. The other three records of patients with aortic regurgitation showed considerable spread, overlapping with those of the normotensive and hypertensive patients. The dicrotic notch (incisura), which was usually well marked in most brachial records of patients with aortic regurgitation, always appeared at a lower point on the catacrotic limb in these subjects than in those of the control and hypertensive group. However, we were unable to associate a low dicrotic notch with a low diastolic blood pressure or the height of the pulse pressure, indicating that the position and characteristics were probably influenced largely by the peripheral factors which alter plus contours.

DISCUSSION

The accuracy of the method of measuring blood pressure by arterial puncture with a small needle and by optical recording with a high frequency manometer seems to have been generally accepted. It effectively overcomes the objection against cannulation methods of blood pressure measurement, as discussed by Wolf and Bonsdorff[4] and Recklinghausen.[2]

Little apparent change in blood pressure resulted from insertion of the needle into the artery, for cuff measurements on the same arm with the needle in situ showed the same values as before insertion. In agreement with Ragan and Bordley,[7] we noted during deflation of the cuff that the Korotkow sounds appeared at the same moment as the initial manometric pulsations from the artery. Wolf and Bonsdorff[4] felt that considerable differences in pressure might occur between these events.

In these experiments, simultaneous graphic recordings were not made from the femoral and brachial arteries because of technical difficulties. Moreover, the blood pressure readings by the cuff method were not made at the same moment as the graphic ones. However, cuff and direct measurements of blood pressure were always made on the same extremity, whereas previous investigators have used one extremity for direct readings and the opposite for indirect measurements. Appreciable differences may exist between cuff measurements from the two arms. Any unusual weighting of results was believed to have been avoided by making graphic measurements alternately, as follows: first in the arm of one subject and first in the leg of the succeeding one. The relatively small difference in pulse rates between the times of making direct measurements in the arm and leg indicates that little change in cardiovascular function occurred to influence the reliability of the comparison of such values.

In agreement with others who compared the accuracy of the auscultatory method with direct measurements in the arm, we found no consistency in the direction of error, either in the systolic or

diastolic levels; the auscultatory readings were sometimes too high, and sometimes too low. However, the averaged errors permit the conclusion that, for general clinical purposes, the auscultatory technique is adequate for the brachial systolic level, provided results in any given case are not interpreted too rigidly. No particular difference in degree of accuracy was found in brachial measurements of systolic pressures in the control or hypertensive groups, or in patients with aortic regurgitation. The diastolic cuff pressures were ascertained less accurately in the arm. Values taken when the arterial sounds changed from sharp to muffled (between third and fourth phases) were generally too high, and the error was of the same degree in the normotensive and hypertensive groups. As one would expect, the error was greater in aortic regurgitation, in which the sound changes are often less marked. Since, to simplify the technique of puncture of the brachial artery, we purposely excluded subjects with large arms, we do not feel that this source of error in cuff measurements enters into the differences.

The standard cuff (13 cm.) technique for femoral pressure was found to be deceptive in almost all subjects; it gave readings which were grossly too high. The wide cuff (15.5 cm.) pressures generally agreed better with direct pressures, although there was still considerable error in many measurements; this error was most marked in patients with aortic regurgitation. Since, in general, the graphic systolic blood pressures in the leg tended to be only slightly higher than those in the arm, the larger differences obtained by either cuff are therefore inferred to be artifacts. The greater mass of soft tissue to be compressed, as well as other anatomic differences, seems to be responsible, for more accurate results can be obtained by a better fitting, wide cuff.

The width (12 to 13 cm.) of the present standard arm cuff is based on the observations of Recklinghausen[2] and Janeway,[13] who recognized that the soft tissue distorts the actual pressure applied to an artery when a narrower cuff (such as Riva-Rocci's) is used. Their choice of the 13 cm. cuff for use on the arm was based on their observation that, with it, approximately the same pressures could be obtained in the leg as in the arm; we could not confirm this observation. For extreme cases they found a wider (15 cm.) cuff adequate for any extremity. Recently, Hamilton and his associates, using children as subjects, and Ragan and Bordley, using adults, recognized the errors from the use of arm cuffs of improper width, and showed that, in general, the width of the cuff should be proportionate to the circumference of the arm. Murray[14] was the first (1914) to report the use of a wider (17 cm.) cuff for femoral pressures. Since then wider cuffs have often been recommended for use on the thigh, but, thus far, there have been no attempts to test their accuracy by comparison with a valid direct method.

The concept that the systolic pressure is higher in the femoral artery in aortic regurgitation originated in 1909 with Hill, et al.,[15] who observed by the palpatory method, with the cuff wrapped around the calf, marked elevation of this pressure over the pressure in the arm. The difference was not limited to aortic insufficiency, for similar discrepancies were noted after excitement and exercise in normal subjects. Murray confirmed this observation, using the 17 cm. cuff. Since then the clinical concept has grown that a higher systolic pressure in the leg is a characteristic, almost specific, sign in aortic regurgitation. In fact, Loewenberg[16] stated that the differentiation between "relative" and true aortic regurgitation may be made on the absence or presence of this sign. This appears to be erroneous because the usual leg cuff will distort the true pressures, and because the true femoral systolic blood pressure is not universally or exclusively higher in definite aortic regurgitation.

Many of the earlier investigators believed that the systolic blood pressure was the same in the arm and leg in normal subjects. On this assumption von Recklinghausen and Janeway based their philosophy of clinical sphygmomanometry. However, Bazett,[17] using one of the cruder membrane manometers, showed that the systolic pressure in the femoral artery was slightly higher than that in the carotid artery in normal dogs. When aortic regurgitation was artificially produced in these animals, the difference was immediately exaggerated, and grew more marked the greater the aortic leak. Burdick and his co-workers,[18] using a 20 cm. cuff for leg measurements, found the femoral systolic pressure moderately higher than the brachial in normal human subjects. Several theories have been advanced to explain these differences in normal subjects and patients with aortic regurgitation. Hürthle[19] believed that the higher systolic pressure in the femoral artery was due to summation of the primary pulse wave and reflected waves from the periphery and from other neighboring vessels. L. Hill and his co-workers[15] attributed the difference to better conduction of the pulse wave into the femoral vessels and to decreased distensibility of the femoral artery. Bazett[17] felt that there was greater conversion of kinetic energy into stress in the femoral artery because of regional differences. Bramwell and Hill[20] believed that "breaker" phenomena at the peak of the onrushing wave front of the pulse were responsible for the greater difference in aortic regurgitation. It is likely that a combination of these factors is responsible.

An attempt was made to correlate the error in the arm cuff readings with the contour of the graphic curves, but no consistent traits in this limited number of subjects were noted. In general, however, the steeper the pulse upstroke in patients with hypertension and aortic regurgitation, the more often the systolic cuff pressures were equal to, or lower than, the direct values. The accuracy of the arm cuff readings was also compared with the Q-E interval (time from onset of

electrical systole to beginning of the upstroke of the brachial pulse curve). As a rule, the Q-E interval was shortest in hypertensive patients (.15 to .18 second), of intermediate duration in normal subjects (.20 to .23 second), and in patients with aortic regurgitation it varied considerably (.15 to .25 second). Since the Q-E interval was essentially the same for the arm and leg at the points where pressure measurements were made, a difference in the velocity of the pulse wave appears not to be a factor in differences between brachial and femoral blood pressures.

SUMMARY

A comparison has been made of the blood pressures measured in the arm and leg with the cuff and usual auscultatory technique and those obtained by direct methods with the needle manometer. The ordinary arm cuff (13 cm. in width) was used for the arm, and this same cuff and a wider (15.5 cm.) one, such as that recommended by the American Heart Association and the Cardiac Society of Great Britain and Ireland, were used for measurements of femoral pressures. Subjects with normal blood pressure, hypertension, and aortic regurgitation were studied.

The cuff pressures in the arm varied both above and below the direct values. As a group, the error was slight for the systolic level, averaging +0.5 per cent, but reached more sizeable proportions for the diastolic pressures of the normotensive and hypertensive groups, in which it was of the order of + 9 per cent. The diastolic cuff error was even greater in subjects with aortic regurgitation, averaging +24 per cent.

The femoral pressures obtained with the 13 cm. cuff on the thigh were grossly above the systolic and diastolic values recorded by the direct method (averaged systolic/diastolic error, normotensive group, $\frac{+17 \text{ per cent}}{+58 \text{ per cent}}$ hypertensive group $\frac{+28 \text{ per cent}}{+46 \text{ per cent}}$; and subjects with aortic regurgitation $\frac{+34 \text{ per cent}}{+92 \text{ per cent}}$). The 15.5 cm. cuff permitted more accurate approximation of the true systolic pressure in the normotensive and hypertensive groups (averaged error, +2.6 per cent, +8 per cent, respectively), but here also the results are in error for the diastolic pressure, averaging +25 per cent for the combined groups. The systolic and diastolic values with the wide cuff on the thigh were most in error in subjects with aortic regurgitation (averaging +29 per cent and +67 per cent, respectively).

The direct femoral systolic blood pressure was notably higher than the direct brachial systolic pressure in five of the nine normotensive subjects, four of the nine hypertensive patients, and seven of the ten patients with aortic regurgitation. In the other cases the difference was slight, although in a few cases the systolic pressures were decidedly

lower in the femoral artery than in the brachial. The limited number of cases and lack of uniformity of results do not permit any definite conclusion. However, it may be pointed out that the femoral systolic pressure was more frequently elevated in the patients with aortic regurgitation, and that the differences were usually greater here than in the control and hypertensive groups.

CONCLUSIONS

1. The systolic blood pressure in the arm can be measured with reasonable accuracy in most subjects with normal pressure, hypertension, and aortic regurgitation by the ordinary arm cuff and auscultatory technique.

2. The brachial diastolic pressure, as measured by the ordinary cuff, is usually too high, especially in aortic regurgitation.

3. The femoral systolic blood pressure cannot be measured accurately with the ordinary 13 cm. cuff. A wider (15.5 cm.) cuff permits more accurate measurement of the femoral systolic pressure, except in subjects with aortic regurgitation whose pressures cannot be measured accurately with either cuff.

4. Femoral diastolic pressures obtained with either cuff were grossly inaccurate in all subjects.

5. The difference between the blood pressure in the arm and leg in patients with aortic regurgitation is not so marked as is generally believed because the cuff, wide or narrow, does not allow true measurements of femoral pressure. Therefore, it is probable that no diagnostic value should be attributed to this sign.

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THE CARDIAC CHILD IN A SPECIAL SCHOOL

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ONE of the major public health problems concerned with children is that of heart disease which is usually one of the sequelae of rheumatic fever. Although an estimate of the prevalence of rheumatic heart disease is difficult to obtain, Paul[1] states that such an estimate in the United States "would indicate that between 350 and 700 cases exist for every 100,000 people." It is well known that not all attacks of rheumatic fever lead to heart disease. This is a point which many feel has been frequently overlooked. Nevertheless, Swift[2] states: "The seriousness of rheumatic fever to the individual and to large groups of individuals needs no arguing, but there has been less emphasis on the number of patients who have recovered from an acute heart attack without heart involvement, and on the possibility of leading a long, useful and happy life, even wth a certain degree of distortion of the heart valves, if the patient has permanently subdued the rheumatic infection. It would thereby seem only the part of wisdom for society to provide the machinery whereby more patients may be moved to this more fortunate class, even though the rheumatic fever still exists. This would be the objective of an efficient health program."

Although much less frequent than the rheumatic type, other forms of heart disease, such as the congenital type, may also cripple many children. An adequate health program is concerned with educating the child's parents and instructing his teachers, and many authorities agree with Connor[3] that: "A proper regime can be instituted and carried out only by the cooperation of the school teachers, the parents, the physician, the school nurse, the medical social worker, the welfare agencies, housing authorities, and departments of health."

In order to form an estimate of the advantages of sending cardiac children to special schools where, in addition to the normal educational program, emphasis is also placed on instructing the child to live and work with his handicap, we have undertaken a study of cardiac pupils who have attended the Jesse Spalding School of Chicago. The Spalding School is one of four elementary schools (first to eighth grades) and one high school set up by the Public School System of the City of Chicago in 1899. These schools were designed to furnish normal school environment and education for handicapped children, and, as designed, were originally intended to assist crippled children to attend school, primarily

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TABLE I

OBSERVATIONS ON 186 CHILDREN WHO ATTENDED SPALDING SCHOOL*

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
140	M	12	Recent chorea	E	½	Rh. M. I.	I	No further special school care felt to be necessary	12	Rh. M. I. and S.	IIA	X			1		Soliciting—light manual	Horseback riding swimming fishing
133	M	15	Recent rheumatic fever and chorea	E	2	Rh. M. I.	I	Graduated—high school	4	No cardiac pathology‡	E	X					Office work odd jobs attending college	
108	M	10	Potential	F	1	Potential	F	No further special school care felt to be necessary	16	No cardiac pathology	F			X			Odd jobs for family—furnace, etc.	Movies
50	M	10	Chorea	F	2	No organic lesion	F	No further special school care felt to be necessary	12	Rh. M. I. (well-comp.)	I	X					Factory—heavy for 8 yr. Factory—light for 2 yr.	Baseball, dancing, football

2	F	13	Potential	F	3	Potential	F	No further special school care felt to be necessary	10	No cardiac pathology	F	X			2 (1 is questionable)	Pregnancy	Light housework	Knitting, sewing, reading, radio, and cards
190	F†	12	Potential	F	3	Potential	F	No further special school care felt to be necessary	5	No cardiac pathology	F	X					Clerical	Dancing skating all athletics
43	F	14	Mitral "regurgitation"	I	½	Mitral "regurgitation"	I	No further special school care felt to be necessary	15	Rh. M. I. and S. (well-comp.)	I	X				Pregnancy	Housework cooking	Movies cooking
1	M	14	Rheumatic fever	I	1	Rh. M. I.	I	No further special school care felt to be necessary	3	No cardiac pathology	F	X					College photography	Photography
156	F	12	Mitral stenosis and chorea	I	1	Potential	F	No further special school care felt to be necessary	15	Mitral "regurgitation" comp. myocardium‡	IIB	X				Pregnancy	Helping in home	Movies, cards, and reading
230	M	10	Rh. M. I.	I	3	Rh. M. I.	I	No further special school care felt to be necessary	17	Rh. M. I.	I		X			Accident	Heavy labor	Baseball bowling
124	F	9	Mitral insufficiency	I	5½	Congenital Pat. D. A.	I	Working	7	Congenital Pat. D. A.	I	X					Light factory work	Skating swimming
20	M	15	Rh. M. I.	IIA	½	Rh. M. I. and S.	I	No further special school care felt to be necessary	12	Rh. M. I. and S. (well-comp.)	I			X			Heavy work 1 yr., light work 2 yr., both work for 2 yr.	Baseball swimming

*Three groups (Out of Town, Unable to Locate, No Cooperation) on whom no follow-up physical signs are available are omitted for the sake of brevity.

†Patients who received psychiatric interview.

‡Examination made by family physican.

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
24	M	13	Mitral stenosis	IIA	½	Mitral stenosis	IIA	No further special school care felt to be necessary	12	No cardiac pathology	F	X				"Flu"	Light assembling selling inducted into Army	Baseball
96	M	14	Rh. M. I. and S.	IIA	½	Rh. M. I. and S.	I	No further special school care felt to be necessary	9	Rh. M. I.	I	X				Pneumonia "strep" throat	Light work college	Baseball
139	M	13	Rh. M. I.	IIA	½	Rh. M. I.	I	No further special school care felt to be necessary	3	Rh. M. I. (well-comp.); paroxysmal tachycardia	IIA	X				Attacks of tachycardia	Barber school	Sports radio
152	F	13	Mitral stenosis	IIA	½	Mitral stenosis	IIA	Moved	14	Mitral stenosis	I	X				Appendicitis pregnancy	Clerk light housework	Bowling movies reading
208	M	14	Congenital heart disease	IIA	½	Congenital heart disease	IIA	Moved	5	Congenital heart disease	III		X			Frequent coughs and colds	Light clerical	Reading

6	F	14	Rh. M. I. and S.	IIA	1	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	6	Rh. M. I. and S.‡ A. I. and S.	IIB	X				Pregnancy	Cashier, part time	Movies
40	F	16	Rh. M. I. and S.	IIA	1	Rh. M. I. and S.	IIA	Ill at home	6	Rh. M. I. and S.	IIA	X				Appendectomy	Housework	Reading girl's club
57	F	15	Rh. M. I. and S.	IIA	1	Rh. M. I. and S.	IIA	Overage—staying at home	5	Rh. M. I. and S.	IIA	X				"Home in bed" "nervous"	Light factory work	Movies
61	F	14	Rh. M. I. and S.	IIA	1	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	4	Rh. M. I. (well-comp.)	I	X				Asthma appendicitis	Secretarial	Dancing swimming
207	F	6	Mitral insufficiency	IIA	1	Rh. M. I.	I	No further special school care felt to be necessary	12	Mitral insufficiency‡	I	X				Pregnancy	Housework	Movies
117	M	11	Rh. M. I.	IIA	1	Rh. M. I.	IIA	No further special school care felt to be necessary	15	Rh. M. I. A. I.	IIB	X			1		Barber	Short wave radio operator radio school
122	M	14	Mitral insufficiency	IIA	1	Potential	F	No further special school care felt to be necessary	13	Potential	F	X				Pneumonia 4 times	Truck driver	Roller skating
145	F	12	Rh. M. I.	IIA	1	Rh. M. I.	IIA	No further special school care felt to be necessary	10	Rh. M. I. A. I.	IIA	X				2 pregnancies mastoidectomy	Own housework	Movies auto rides
222	M	15	Rh. M. I.	IIA	1	Rh. M. I.	IIA	No further special school care felt to be necessary	12	Rh. M. I. and S.	IIA	X				Occasional colds	Light work	Reading, baseball, and ice skating

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
225	F	15	Rh. M. I.	IIA	1	Rh. M. I.	IIA	Dismissed—pregnant	9	Rh. M. I.	I	X			2	Sinus pregnancy	Housework	Reading
13	F	17	Rh. M. I. and S. A. I.	IIA	1	Rh. M. I. and S. A. I.	IIB	Overage—staying at home	3	Rh. M. I. and S. A. I.	III	X			5	Fibrillating 2 yr.	Light housework	Girl's club
22	F	13	Congenital heart disease; Pat. D. A.	IIA	2	Congenital heart disease; Pat. D. A.	IIA	No further special school care felt to be necessary	15	Possible heart disease; old Pat. D. A.	E	X				U.R.I. hemorrhoidectomy	Typist	Movies reading
29	F	14	Rh. M. I. and S.	IIA	2	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X					Light factory work	Dancing
52	M	9	Congenital Pat. D. A.	IIA	2	Congenital Pat. D. A.	I	No further special school care felt to be necessary	13	Congenital —Pat. D. A.; possible interventricular defect of the septum	I	X				Occasional cold	Traveling salesman	Golf basketball
47	F	8	Rh. M. I. and S.	IIA	2	Rh. M. I. and S.	IIA	To parochial school	16	Rh. M. I and S.	IIB	X			Twice decompensated	2 pregnancies 2 cesarians	Housework	Movies visiting

63	F†	13	Rh. M. I. and S.	IIA	2	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	5	Rh. M. I and S.	IIA		X				Light housework	Dancing embroidery
58	F	16	Rh. M. I.	IIA	2	Rh. M. I.	I	No further special school care felt to be necessary	6	Rh. M. I.	I	X				3	Miscarriage	Housework	Reading
111	M	15	Rh. M. I. and S.	IIA	2	Rh. M. I. and S. A. I.	IIA	Working	3	Rh. M. I. and S. A. I.	IIB	X				1	Pneumonia	Light factory work (standing)	Dancing
218	F	14	Congenital	IIA	2	Congenital	IIA	Working	3	Congenital—patent interventricular septum	I	X						Housework	Music (voice) sewing
146	M	14	Rh. M. I. and S.	IIA	2	Rh. M. I.	IIA	No further special school care felt to be necessary	12	Rh. M. I. and S.	IIA	X					Pleurisy tonsillectomy	Light factory	Baseball photography
227	M	14	Rh. M. I.	IIA	2	Rh. M. I.	I	No further special school care felt to be necessary	4	Possible heart disease	E			X				Light work in printing office	Violin, art work, and bowling
102	M	14	Rh. M. I. and S. with arrhythmia	IIA	2	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X					Leg injury	Light office work	Fishing swimming
49	F	15	Rh. M. I. and S. with extra systoles	IIA	2	Rh. M. I. and S. with extra systoles	I	No further special school care felt to be necessary	4	Rh. M. I. and S. with extra systoles	IIA			X				Light office and housework	Movies dancing

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
177	F	14	Rh. M. I. and S.	IIA	2	Rh. M. I. and S.	IIA	Overage—staying at home	4	Rh. M. I. and S.	IIA	X				Pneumonia	Light factory work	Dancing baseball
158	M	9	Rh. M. I.	IIA	2	Rh. M. I.	IIA	No further special school care felt to be necessary	8	Rh. M. I.	I	X					Attends optometry school	Reading music
217	F	13	Congenital	IIA	2	Congenital	IIA	No further special school care felt to be necessary	3	Congenital heart disease, Pat. interventricular septum	IIB	X				2 "nervous" attacks	Secretarial	Dancing movies
148	F	10	Rh. M. S.	IIA	2	Rh. M. S.	IIA	To parochial school	12	Rh. M. I. and S.	IIB	X					Office work	Church movies
219	F†	11	Rh. M. I. and S.	IIA	3	Rh. M. I. and S.	IIA	Left against medical advice	5	Mitral stenosis and "regurgitation"‡	IIA	X				Pregnancy	Light office work part-time waitress	Reading
42	F†	12	Post-diphtheritic heart disease	IIA	3	Post-diphtheritic heart disease	IIA	No further special school care felt to be necessary	5	Mitral and aortic insufficiency and stenosis; Pat. D. A. post-diphtheria and scarlet fever	IIB	X				"Strep" throat, appendectomy	Waitress	Dancing sewing

155	F	16	Aortic "regurgitation"	IIA	3	Aortic "regurgitation"	IIA	Graduated—high school	9	Rh. M. I. and S. A. I. and S.	IIB	X			1	Auto accident	Office work	No time
35	F†	13	Rh. M. I. and S.	IIA	3	Rh. M. I. and S.	I	No further special school care felt to be necessary	4	Rh. M. I.	I	X					Light factory work	Swimming
16	M	12	Rh. M. I. and S.	IIA	3	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	6	Rh. M. I.	I	X					Heavy labor light labor	Mechanics
10	M†	12	Chorea	IIA	3	Potential	F	No further special school care felt to be necessary	5	No cardiac pathology	F	X					Odd jobs college	Mechanics swimming
38	M†	11	Rh. M. I.	IIA	3	Rh. M. I.	IIA	No further special school care felt to be necessary	5	Rh. M. I. and S. A. I. and S.	I	X					Window decorator	Speed boating
79	F	14	Rh. M. I.	IIA	3	Rh. M. I.	IIA	No further special school care felt to be necessary	7	Rh. M. I. (well-comp.)	IIA		X		1		"26" girl	Dancing
126	M	14	Rh. M. I. and S.	IIA	3	Rh. M. I. and S. (well-comp.)	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X				Sore throat	Light factory work	Baseball
178	F	11	Rh. M. I. and S.	IIA	3	Rh. M. I. and S.	IIB	Moved	13	Rh. M. I. and S.	I	X				Pregnancy	House-work	Movies
221	M	16	Rh. M. I. and S.	IIA	3	Rh. M. I. and S. A. I.	IIB	Working	2	Rh. M. I. and S. A. I. and S.	IIA	X					Light factory work	Baseball
103	F†	14	Rh. M. I. and S.	IIA	4	Rh. M. I. and S.	IIA	Graduated—high school	3	Rh. M. I. and S.	IIA	X					Secretarial	Sewing cooking

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
199	M	11	Rh. M. I.	IIA	4	Rh. M. I.	IIA	No further special school care felt to be necessary	6	Rh. M. I.	I	X					Drug-store clerk school	Y.M.C.A. swimming
170	F	14	Mitral insufficiency	IIA	4	Mitral insufficiency	IIA	Overage—staying at home	3	Mitral insufficiency (comp.)	I	X			2	Glaucoma	Light factory work	Baseball swimming
162	F	10	Rh. M. I. and S.	IIA	4	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X					School	Dancing
100	F†	16	Mitral insufficiency	IIA	5	Mitral insufficiency	I	Overage—staying at home	7	Rh. M. I. and S. A. I. and S.	IIB	X				Mild thyrotoxicosis, pregnancy	Housework	Reading
198	F†	9	Rh. M. I. and S.	IIA	5	Rh. M. I. and S. (well-comp.)	IIA	No further special school care felt to be necessary	7	Rh. M. I.	I	X				Pregnancy	Office work	Social life
189	M	12	Rh. M. I. and S.	IIA	5	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	1	Rh. M. I.	IIA	X					School	Baseball swimming
83	M	10	Rh. M. I. and S.	IIA	5	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	1	Rh. M. I.	I	X					School professional golfing	Clarinet golf

98	M	14	Rh. M. I.	IIA	5	Rh. M. I. A. I.	IIA	Graduated—high school	4	Rh. M. I. and S. A. I.	I	X				Tooth abscess	Tool and die maker	Horseback riding
107	F	8	Rheumatic mitral "regurgitation"	IIA	5	Rheumatic mitral "regurgitation"	I	No further special school care felt to be necessary	9	Possible heart disease	E	X				Ovarian cyst	Housework	Dancing
37	M	12	Rh. M. S.	IIA	5	Rh. M. I. and S.	IIB	Working	9	Rh. M. I. and S. with extra systoles	IIA	X				Taking digitalis	Barber	Bowling
70	F	12	Rh. M. I.	IIA	5	Rh. M. I.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S. A. I.	IIA		X			Sore throat	Light factory work	Dancing
80	F	6	Rh. M. I. and S.	IIA	5	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S. A. I. and S.	IIB	X			1		Light factory work	Reading knitting
45	F	13	Rh. M. I. and chorea	IIA	5	Rh. M. I.	IIA	Working	6	Rh. M. I. and S.	IIA	X				Sore throat	Light factory work	Dancing
187	F	13	Rh. M. S.	IIA	6	Rh. M. S.	IIA	Ill at home	9	Rh. M. I. and S.	IIB	X				"Flu"	Office work	Fond of the country
78	F	8	Mitral insufficiency	IIA	7	Mitral insufficiency and stenosis	IIA	No further special school care felt to be necessary	8	Mitral insufficiency	I	X					Light factory work	Music beauty culture
88	F†	17	Paroxysmal tachycardia; possible congenital	IIA	3	Paroxysmal tachycardia; possible congenital	III	Ill—hospitalized	6	Paroxysmal tachycardia, Pat. D. A.	I	X					Teaches music at home	Piano

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
161	M	8	Rheumatic mitral "regurgitation"	IIA	8	Rh. M. I. and S.	IIB	No further special school care felt to be necessary	10	Rheumatic heart disease; auricular fibrillation	III	X			2			Photography
90	F	8	Rh. M. I. and S.	IIA	8	Rh. M. I. and S. A. I.	IIA	No further special school care felt to be necessary	4	Rh. M. I. and S. A. I. and S.	IIA	X					Sedentary assembling	Roller skating
123	F	12	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	IIB	Transferred—distance to special school too great	9	Rh. M. I. (well-comp.)	I		X			Pregnancy U.R.I.	Housework outside of home	Dancing, ice skating, and swimming
194	F	16	Rh. M.I. and S. A. I. and S.	IIB	1	Rh. M. I. and S. A. I. and S.	IIA	Working	13	Rh. M. I. A. I.	IIA	X			1	2 full-time pregnancies, 2 miscarriages	Housework care of children	Roller skating
137	M	10	Mitral stenosis	IIB	1	Mitral stenosis	IIA	No further special school care felt to be necessary	18	Rh. M. I. (well-comp.)	I	X				"Bronchial trouble" needs x-ray	Heavy labor	Tumbling boxing

54	F	11	Rheumatic heart disease chorea	IIB	1	Rheumatic heart disease	IIA	No further special school care felt to be necessary	12	Rh. M. I.	I	X					Housework	Walking
101	M	15	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	10	Rh. M. I. and S. A. I. and S.	IIB	X				Stomach trouble	Heavy labor	Photography
15	M	13	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	IIB	No further special school care felt to be necessary	18	Rh. M. I. and S.	I	X				Operation	Light work	Photography
19	M†	16	Rh. M. I. and S. A. I.	IIB	1½	Rh. M. I. and S. A. I.	IIB	Left against medical advice	7	Rh. M. I. and S. A. I.	IIA	X				Occasional cold	Light work	Handy about the house
81	M	16	Rh. M. I. and S.	IIB	2	Rh. M. I. and S.	IIB	Dismissed—municipal tuberculosis sanitarium. (Pulmonary tuberculosis)	6	Rh. M. I.‡	IIA	X				Dementia praecox	None	None
209	M	14	Congenital interventricular septal defect	IIB	2	Congenital interventricular septal defect	IIA	No further special school care felt to be necessary	6	Congenital	IIA	X					Light work	Bowling
165	M	17	Rh. M. I. and S. A. I.	IIB	2	Rh. M. I. and S. A. I.	IIB	Ill at home	6	Rh. M. I. and S. A. I.	IIB	X				Occasional cold and sore throat	Light work	Stamp collecting bowling
71	M	12	Rh. M. I. and S.	IIB	2	Rh. M. I. and S.	IIB	No further special school care felt to be necessary	18	Rheumatic pancarditis; decompensation; auricular fibrillation‡	III	X			1	In the hospital at the time	Traveling salesman	Not known

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
134	M	16	Hypertension, cardiorenal, vascular type	IIB	2	Hypertension cardiorenal vascular type	IIB	No further special school care felt to be necessary	7	Hypertension	E	X					Light work	Sports reading
188	M	14	Rh. M. I. and S. A. I.	IIB	2	Rh. M. I. and S. A. I.	IIB	No further special school care felt to be necessary	5	Rh. M. I. and S. A. I. and S.	IIB	X			2	"Flu"	Light work	Reading
25	F	13	Rh. M. I. A. I.	IIB	3	Rh. A. I.	IIA	No further special school care felt to be necessary	5	Rh. A. I.	IIA	X					School	Dancing reading
30	F	12	Rh. M. I. and S. A. I.	IIB	3	Rh. M. I. and S. A. I.	IIB	No further special school care felt to be necessary	2	Rh. M. I. and S. A. I. and S.	IIA	X					Light work	Sewing beauty culture
82	M	15	Congenital	IIB	3	Congenital	IIB	Working	15	Congenital	IIB	X				"Strep" infection pleurisy	Heavy work	Movies reading
87	M	15	Rh. M. I. and S. A. I. and S.	IIB	3	Rh. M. I. and S. A. I. and S.	IIB	Working	4	Rh. M. I. and S. A. I. and S.	IIA	X			2		Heavy work	Art work baseball

95	F	16	Rh. M. I. and S.	IIB	2	Rh. M. I. and S.	IIA	Working	7	Rh. M. I. and S. (well-comp.)	I	X				Appendectomy	Housework	Swimming
114	F†	14	Rh. M. I.	IIB	3	Rh. M. I.	IIA	Overage—staying at home	7	No cardiac pathology‡	F	X				4 pregnancies	Housework	Movies
136	F	13	Rh. M. I. and S.	IIB	3	Rh. M. I. and S.	IIB	Ill—hospitalized	5	Rh. M. I. and S.	IIA	X			10	Operation	None	Handwork
169	M	15	Rh. M. I. and S.	IIB	3	Rh. M. I. and S.	IIB	Moved	6	Rh. M.I. and S.; A. I.	IIA	X				Psychotic	Light work	Photography
173	M	15	Rh. M. I. and S. A. I. and S.	IIB	3	Rh. M. I. and S. A. I. and S.	IIB	No further special school care felt to be necessary	4	Rheumatic heart disease; auricular fibrillation	IIB	X			2		Light work	Printing
184	M	14	Rh. M. I. and S. A. I. and S.	IIB	3	Rh. M. I. and S. A. I. and S.	IIB	Left against medical advice	5	Rh. M. I. and S. A. I.	IIA	X					Light work	Assistant scoutmaster dancing
213	F	11	Rh. M. I. and S.	IIB	3	Rh. M. I. and S.	IIA	Graduated—high school	14	Rh. M. I. and S.	IIA	X					High school	Baseball hockey swimming
17	F	12	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	IIB	Graduated—high school	3	Rheumatic heart disease; auricular fibrillation	III	X			2		School	Dancing
77	F†	11	Congenital patent interventricular septum	IIB	4	Congenital Pat. D. A.	IIB	No further special school care felt to be necessary	5	Congenital, Pat. D. A.	I	X				Accident	Light work	Piano dancing

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
105	M	10	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. (well-comp.)	I	X			2		School light work	Baseball singing
110	F	11	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X				"Flu"	School light work	Swimming
171	F	11	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	4	Rh. M. I. and S.	IIA	X					School light work	Singing
172	M	14	Rh. M. I. and S. A. I.	IIB	4	Rh. M. I. and S. A. I.	IIA	Graduated—high school	2	Rh. M. I. and S. A. I.	IIA	X					School light work	Bowling
23	M	11	Rheumatic mitral "regurgitation"	IIB	4½	Rheumatic mitral "regurgitation"	IIA	No further special school care felt to be necessary	4	Rh. M. I. with extra systoles	IIA	X				Overdigitalization	School	Photography
138	M	12	Rheumatic endocarditis	IIB	4½	Rheumatic endocarditis	IIB	No further special school care felt to be necessary	9	No cardiac pathology‡	F	X				Occasional tachycardia	Not known	Not known
31	F	11	Rh. M. I. and S.	IIB	5	Rh. M. I. and S.	IIA	Graduated—high school	2	Rh. M. I. and S.	IIA	X			1 (in bed 1 yr.)	Taking digitalis now	None	Movies

66	M	13	Rh. M. I. and S.	IIB	5	Rh. M. I. and S.	IIA	Graduated—high school	4	Rh. M. I. and S. (well-comp.)	I	X					Medium work	Fishing
113	F	10	Congenital with superimposed Rh. I. and S.	IIB	5	Rh. M. I. and S.	IIA	Overage—staying at home	4	Rh. M. I. and S. A. I.	IIA	X				Cyst operation	Light work	Dancing
128	M†	14	Rheumatic auricular fibrillation	IIB	6	Rh. M. I. and S.	IIB	Graduated—high school	6	Rh. M. I and S. A. I.	IIA	X				Nasal operation	Light work	Reading
151	F	13	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIA	Graduated—high school	4	Rh. M. I. and S. A. I. and S.	IIB	X			2 (in bed 1 yr.)	Pneumonia, accident	Housework	Radio cooking
159	M†	12	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	5	Rh. M. I.	I	X				Appendicitis, hernia	Light work	Reading radio
181	M†	16	Rh. A. I.	IIB	2	Rh. A. I.	IIB	Working	7	Rh. M. I. and S. A. I.	IIA	X			1		Medium work	Stamp collecting bowling
220	M	11	Rheumatic mitral "regurgitation" and stenosis	IIB	6	Rheumatic mitral "regurgitation" and stenosis	IIB	Overage—staying at home	3	Rh. M. I. and S. A. I.	IIB	X					Light work	Baseball
55	M	11	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	1	Rh. M. I. (well-comp.)	I	X					School	Baseball

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	ON RECHECK PHYSICAL EXAMINATION		MEDICAL CARE			RHEUMATIC RECURRENCES	CARDIAC EPISODES AND/OR OTHER ILLNESSES	JOBS HELD	RECREATION
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION			DIAGNOSIS	CLASSIFICATION	WITHIN 3 YEARS	MORE THAN 3 YEARS AGO	NONE				
67	F	14	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIB	Graduated—high school	6	Rheumatic heart Disease‡	IIB	X				Pregnancy	House-work	Sewing
176	F	8	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	1	Rh. M. I. and S.	IIB	X			1		School	Piano
180	M	11	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	1	Rh. M. I. and S. A. I. and S.	IIB	X					Trade school	Accor-dion baseball
206	M	10	Rh. M. I. and S. A. I.	IIB	6	Rh. M. I. and S. A. I.	IIA	No further special school care felt to be necessary	1	Rh. M. I. and S. A. I.	IIA	X				Pneumo-nia	Light work	Baseball
231	F	10	Rh. M. I. and S.	IIB	6	Rh. M. I. and S.	IIB	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIB	X				T. & A.	Light work	Tennis
182	F	8	Congen-ital	IIB	7	Congen-ital	IIB	No further special school care felt to be necessary	9	Congenital	IIB	X				Dizziness	Light work	Movies radio

12	M	8	Rh. M. I. A. I.	IIB	7	Rh. M. I. and S. A. I.	IIB	Moved	8	Rh. M. I. and S. A. I. and S.	IIB	X					Medium work	Skiing travel
125	M	9	Rh. M. I. A. I.	IIB	7	Rh. M. I. A. I.	IIB	No further special school care felt to be necessary	5	Rh. M. I. and S. A. I. and S.	IIB	X			1	Colds	Medium work	Movies
232	M	13	Congenital	IIB	7	Congenital	IIB	Graduated—high school	3	Congenital patent interventricular septal defect	IIA	X				"Streptococcal grippe"	School light work	Art work
116	F	8	Rh. M. I. and S.	IIB	8	Rh. M. I. and S.	IIB	Overage—staying at home	3	Rh. M. I. and S. A. I.	IIB	X				Gall bladder attacks	Light work	Dancing movies
46	M	9	Rh. M. I. and chorea	IIB	9	Rh. M. I.	IIA	No further special school care felt to be necessary	3	Rh. M. I. and S.	IIA	X					Trade school	Baseball
167	F	8	Rh. M. S.	IIB	9	Rh. M. I. and S.	IIA	Working	8	Rh. M. I. and S. with extra systoles	IIA	X				"Cyst of back" pregnancy	House-work	Sewing
27	F	10	Rh. M. I. and S.	IIB	10	Rh. M. I. and S.	IIB	Graduated—high school	4	Rheumatic mitral lesion; auricular fibrillation‡	IIB	X			1		Light work	Sewing reading
135	M	7	Rh. M. I. and S.	IIB	11	Rh. M. I. and S.	IIB	No further special school care felt to be necessary	9	Rh. M. I. and S.	IIA	X				U.R.I.	Medium work	Photography
179	M	7	Rh. M. I. and S. A. I. and S.	IIB	11	Rh. M. I. and S. A. I. and S.	IIB	Graduated—high school	7	Rh. M. I. A. I.‡	IIB	X				U.R.I.	Not known	Not known

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
216	F	9	Mitral insufficiency and stenosis	I	4	Mitral insufficiency and stenosis	I	No further special school care felt to be necessary	11	Subacute bacterial endocarditis; rheumatic heart disease		24
132	M	14	Congenital heart disease	IIA	½	Congenital pulmonary stenosis	IIA	Left against medical advice	2	Morbus caeruleus, impotency of foramen ovale		16½
168	F	15	Organic heart disease	IIA	1	Organic heart disease	IIA	No further special school care felt to be necessary	1	Mitral stenosis and regurgitation		17
211	M	9	Mitral insufficiency and stenosis; postscarlatinal	IIA	1	Mitral insufficiency and stenosis; postscarlatinal	III	Ill at home	1			11
174	M	11	Mitral insufficiency and stenosis; aortic insufficiency	IIA	1	Mitral insufficiency and stenosis; aortic insufficiency	IIA	No further special school care felt to be necessary	9			21
112	F†	14	Congenital heart disease; interventricular septal defect	IIA	2	Congenital heart disease; interventricular septal defect	IIA	Overage—staying at home	4	Malignant endocarditis	X	20
147	M†	11	Rh. M. I.	IIA	2	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	3	Chronic myocarditis		16
118	M	10	Rh. M. I. and S.	IIA	2½	Rh. M. I. and S.	IIA	Ill at home	11	Inactive rheumatic heart disease with M. I. and S. A. I. and S.		23½

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
5	M	7	Mitral insufficiency and stenosis	IIA	3	Mitral insufficiency and stenosis	III	Ill at home	2	Chronic myocarditis with ascites		12
9	M	9	Rh. M. I. and S.	IIA	3	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	11			23
64	F	8	Rh. M. I.	IIA	3	Rh. M. I. and S.	IIA	No further special school care felt to be necessary	6	Rheumatic heart disease		17
84	F	14	Mitral insufficiency and stenosis	IIA	3	Rh. M. I. and S. A. I.	IIA	Working	3	Acute bacterial endocarditis		20
56	M	13	Rh. M. I. and S. A. I. and S.	IIA	3½	Rh. M. I. and S. A. I. and S.	IIB	Ill at home	1	Chronic rheumatic endocarditis and myocarditis		17½
183	F	14	Congenital heart disease	IIA	3½	Congenital heart disease	IIA	Working	8			25½
186	F	9	Rh. M. I. and S.	IIA	3½	Rh. M. I. and S.	III	Ill—hospitalized	1	Mitral stenosis and regurgitation; rheumatic endocarditis		13½
215	F	10	Mitral insufficiency and stenosis	IIA	4	Mitral insufficiency and stenosis	IIA	Moved	4	Chronic endocarditis; myocarditis		18
119	F†	14	Rh. M. I. and S.	IIA	4	Rh. M. I. and S.	IIA	Graduated—high school	7	Acute cardiac failure; rheumatic endocarditis	X	25
144	M	7	Mitral insufficiency and stenosis	IIA	4	Mitral insufficiency and stenosis	IIA	No further special school care felt to be necessary	8	Rheumatic heart disease; double mitral and aortic lesions		19

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
212	F	10	Mitral insufficiency and stenosis	IIA	4	Rh. M. I. and S. A. I. and S.	III	Ill—hospitalized	½	Mitral insufficiency and stenosis; cardiac hypertrophy and dilatation		14½
74	M	11	Rh. M. I. and S.	IIA	4½	Rh. M. I. and S.	IIA	Ill at home	9			24½
7	M	10	Mitral insufficiency and stenosis	IIA	7	Rh. M. I. and S.	IIA	Overage—staying at home	2	Rheumatic heart disease		19
8	F	9	Organic heart disease	IIB	½	Organic heart disease	IIB	No further special school care felt to be necessary	2			11½
86	F	8	Rh. M. I.	IIB	½	Rh. M. I. and S.	IIB	Ill at home	5	Chronic valvular heart disease with acute exacerbation; acute articular rheumatism		13½
154	F	15	Rheumatic myocarditis	IIB	½	Rheumatic myocarditis	IIB	Ill at home	1	Rheumatic heart disease with mitral stenosis; regurgitation pericarditis		16½
201	F	15	Rh. M. I. and S. A. I.	IIB	½	Rh. M. I. and S. A. I.	III	Ill at home	1			16½
205	M	15	Rh. M. I. and S.	IIB	½	Rh. M. I. and S.	IIB	Left against medical advice	3			18½
14	M	15	Rh. M. I. and S.	IIB	1	Rh. M. I. and S. A. I.	III	Ill—hospitalized	1	Rheumatic heart disease with double mitral and double aortic		17

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
32	F	16	Rh. M. I.	IIB	1	Rh. M. I. and S.	IIB	Ill at home	1	Terminal lobar pneumonia; chronic rheumatic endocarditis with cor bovinum		18
97	F	14	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	IIB	Ill at home	1	Organic heart disease; acute pulmonary edema		16
99	M†	16	Rh. M. I. and S. A. I.	IIB	1	Rh. M. I. and S. A. I. and S.	IIB	Graduated—high school	4	Subacute bacterial endocarditis with multiple infarcts in kidneys and spleen		21
143	M	13	Congenital heart disease	IIB	1	Congenital heart disease	IIB	Moved	3	Rheumatism endocarditis		17
160	F	13	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	III	Ill at home	3	Decompensation mitral stenosis		17
224	F	9	Rh. M. I. and S.	IIB	1	Rh. M. I. and S.	IIB	Transferred—distance to special school too great	3			13
34	M	13	Rh. M. I. and S. A. I.	IIB	2	Rh. M.I. and S. A. I. and S.	IIB	Moved	2	Chronic endocarditis		17
72	F	13	Rh. M. I. Congenital	IIB	2	Congenital with superimposed Rh. M. I. and S. A. I.	IIB	Left against medical advice	4	Cerebral embolus, endocarditis, and pleurisy		19
129	F	16	Organic heart disease	IIB	2	Organic heart disease	III	Ill at home	Date unknown			

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
157	M	14	M. I. and S. A. I. and S.	IIB	2	M. I. and S. A. I. and S.	IIB	Working	9	Mitral regurgitation; chronic articular rheumatism		25
192	F	9	Mitral "regurgitation"	IIB	2	M. I. and S.	III	Ill at home	2	Valvular heart disease with decompensation; acute endocarditis		13
18	M	8	Rh. M. I. and S.	IIB	3	Rh.M. I. and S.	IIB	Moved	2	Chronic myocarditis		13
68	M	14	Rh. M. I. and S. A. I. and S.	IIB	3	Rh. M. I. and S. A. I. and S.	IIB	Graduated—high school	2	Rheumatic heart disease		19
76	M	14	Rh. M. I. and S.	IIB	3	Rh. M. I. and S.	IIB	Moved	1	Lobar pneumonia		18
202	F	13	Rh. M. I. and S.	IIB	3	Rh. M.I. and S.	IIB	Overage—staying at home	4			20
203	F	14	Mitral insufficiency	IIB	3	Mitral insufficiency and stenosis	IIB	Overage—staying at home	3			20
233	M	12	Congenital heart disease	IIB	3	Congenital heart disease	IIA	No further special school care felt to be necessary	4			19
21	F	12	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	IIB	Ill at home	2	Endocarditis nephritis		18
28	F	13	Congenital heart disease	IIB	4	Congenital heart disease	IIB	Working	2	Chronic myocarditis		19
59	M	16	Rheumatic pancarditis	IIB	4	Rheumatic pancarditis	IIB	Graduated—high school	2	Chronic rheumatic endocarditis; chronic valvular heart disease		20

TABLE I—CONT'D

CODE NUMBER	SEX	ON ENTRANCE TO SPALDING			TOTAL YEARS AT SPALDING SCHOOL	ON LEAVING SPALDING		REASON FOR LEAVING SPALDING SCHOOL	NUMBER OF YEARS AFTER LEAVING SPALDING	CAUSE OF DEATH COPIED FROM DEATH CERTIFICATE	AUTOPSY	AGE AT DEATH
		AGE (YR.)	DIAGNOSIS	CLASSIFICATION		DIAGNOSIS	CLASSIFICATION					
89	F	9	Rh. M. I. and S.	IIB	4	Rh. M. I. and S.	III	Ill—hospitalized	2	Chronic myocarditis with decompensation; pericarditis (acute)		15
210	M	16	Congenital heart disease	IIB	4	Congenital heart disease	IIB	Graduated—high school	4	Rheumatic heart disease		24
69	F†	14	Congenital heart disease	IIB	5	Congenital heart disease	IIB	Graduated—high school	3	Bacterial endocarditis; congenital heart disease		22
121	M	11	Rh. M. I. and S.	IIB	5	Rh. M. I. and S.	III	Ill at home	2	Rheumatic heart disease		18
185	F	15	M. I. and S. A. I. (post-diphtheritic) (post-scarlet)	IIB	5	M. I. and S. A. I. (post-diphtheritic) (post-scarlet)	IIB	Graduated—high school	2	Cerebral embolism; subacute bacterial endocarditis; rheumatic pancarditis	X	22
204	F	12	M. I. and S.	IIB	5	M. I. and S.	IIB	Working	1	Chronic myocarditis; cardiac decompensation		18
85	M	11	Rh. M. I. and S.	IIB	7	Rh. M. I. and S. A. I.	IIB	Graduated—high school	1 mo.	Rheumatic heart disease with auricular fibrillation; cardiac decompensation; adhesive pericarditis	X	18
104	M	9	Congenital heart disease	IIB	7	Congenital heart disease	IIB	Graduated—high school	2	Streptococcic meningitis; congenital heart malformation		18
191	M†	12	Rh. M. I. and S.	IIB	9½	Rh. M. I. and S.	IIB	Graduated—high school	1½	Chronic myocarditis		23

by the use of elevators, ramps, orthopedic equipment, physiotherapy, swimming pools, etc., even if they required constant medical supervision and treatment. In 1924, through the efforts, and under the supervision, of the Chicago Heart Association, cardiac children were admitted to these schools as a group which formed at that time 13 per cent of the total enrollment, and is now substantially increased.

The eligibility of a child to attend one of these special schools depends upon whether he "by reason of disease, accident or congenital deformity, cannot attend a regular school with safety and profit, during the period of his physical rehabilitation, simultaneous mental development, and social adjustment."[4] The Chicago Heart Association appoints a physician for each school, and, in addition, there is full-time nursing service, originally furnished through the facilities of the Visiting Nurse Association of Chicago, and now through the cooperation of the Board of Health of the City of Chicago. Bus transportation, elevators, ramps, and wheel chairs are provided. The nurses take daily pulse rate and temperature readings, carry out all treatments prescribed by private or clinic physicians, conduct supervised rest periods, make home calls to provide some social service contact, and assist the Chicago Heart Association school physician in controlling the activities of the cardiac child in school.

It must be made clear that the medical management in these cases is left entirely to the private or clinic physician. The Chicago Heart Association school physician examines all children referred for admission, and reserves the right to deny admission to noncardiacs, to those with heart disease in Class I who could undertake ordinary school activity, and to those who are too ill for any school activity. On the other hand, he may admit patients with recent infections in order to prevent possible complications and sequelae. In addition, by frequent re-examinations, he limits the activity of the child by rest periods and by controlling the amount of schoolwork and play. He prescribes no medication, but may send the child home or to a hospital, as indicated. Other communities[5] have followed our example with similar plans.

METHOD OF STUDY

All available records of cardiac children who attended Spalding School served as the basis for this study. The recent records were found to be more comprehensive and complete than those of an earlier date, but all provided sufficient information. A total of 233 cases included in the survey falls into five groups:

130 Completely worked up, with recheck physical examinations (Table I).
56 Deceased since leaving school (Table I).
21 Living out of town.
19 Unable to locate.
7 Refused to cooperate.

Thus, 158 are living and presumably well, and 56 are dead. This leaves the small number of 19 unaccounted for. Of the 158 living and well,

130 were available for study. We reviewed their school records, investigated their families and environment, took complete histories of their health since leaving school, and then did our own physical examinations, so that the physical signs that are dependent on subjective interpretation (such as cardiac murmurs) would vary as little as possible. In addition, 32 patients had previously received a psychiatric interview.*

CHICAGO HEART ASSOCIATION

Spalding School

NAME _____ WHITE _____ COLORED _____
MALE _____ FEMALE _____

ADDRESS _____ FL. _____ TELEPHONE _____

BIRTHDATE _____ RELIGION _____ NATIONALITY _____

DIAGNOSTIC SUMMARY

	DIAGNOSIS	AGE	CLASSIFICATION	M.D.	CLINIC NO.
DATE REFERRAL					
DATE ADMISSION					
DATE DISCHARGE					

SCHOOL RECORD

ENTERED SCHOOL	LEFT SCHOOL	ATTENDANCE
DATE _____ AGE _____ GRADE _____	DATE _____ AGE _____ GRADE _____	SCHOOLDAYS ABSENT IN EL.
FROM: _____	TRANS. TO: _____	SCHOOLDAYS ABSENT IN H. S.
NO. SCHOOL YEARS TO DATE:	REASON: _____	TOTAL YRS. IN EL. _____ TOTAL YRS. IN H.S. _____

PSYCHOLOGICAL

DATE OF TEST	CHRON. AGE	MENTAL AGE	IQ	SOURCE

GRADES:
EL. - SUP. _____ EX. _____ GOOD _____ FAIR _____ POOR _____
H.S.- SUP. _____ EX. _____ GOOD _____ FAIR _____ POOR _____

ADJUSTMENT IN SCHOOL _____
COURSE OF STUDY _____
VOCATIONAL CHOICE _____
ACTION TAKEN _____

Fig. 1.—Data primarily on the individual.

The questionnaire used in compiling data consists of five pages:

Page 1. Data primarily on the student (Fig. 1).

Page 2. Family data (Fig. 2).

Page 3. Medical history and results of physical examination on entrance to school, as well as subsequent observations. Here both diagnosis and classification are recorded on first physical examination and upon discharge from school (Fig. 3).

*Conducted by Dr. Paul Schroeder and associates at the Institute for Juvenile Research in Chicago.

Page 4. Current social and economic data secured by home call (Fig. 4).

Page 5. Recheck physical examination done at the office of the Chicago Heart Association by Dr. Arthur F. Abt or Dr. Kate H. Kohn. Twelve recheck physical examinations were done by other physicians because the patients were out of town at the time, or because they were under current care for other medical reasons (Fig. 5).

#2

FAMILY HISTORY

NAME	KIN	AGE	PLACE OF BIRTH	OCCUPATION	WAGE
OTHER MEMBERS IN HOUSEHOLD					

HOME AND NEIGHBORHOOD:

HEALTH OF FAMILY:

ATTITUDE OF FAMILY TOWARD CHILD

FAMILY'S PLAN FOR CHILD

Fig. 2.—Family data.

The Classification of Patients with Diseases of the Heart prepared by the Criteria Committee of the New York Heart Association and adopted by the American Heart Association was used. This classification is referred to throughout this paper because it was used at the time of the early physical examination. It is sometimes referred to as the "first," or "old," classification, as opposed to the "new" one adopted in 1939.[6]

In the course of the study, 494 home calls were made and 520 letters sent.

RESULTS AND DISCUSSION

Two hundred thirty-three cases, the five groups previously mentioned, were studied. There were other cardiac patients in the school during the survey period: thirty-four students who attended for less than one semester, and thirty who died while registered at school; these were not included because their records offered too little information.

MEDICAL #3

HISTORY:
OBTAINED FROM CLINIC OR M.D._____

_____

PREVIOUS HISTORY: _____

_____

ILLNESSES WITH DATES:
SCARLET FEVER _____ TONSILLITIS_____ TONSILLECTOMY___
MEASLES _____ DIPHTHERIA_____ CHOREA_____

HISTORY OF CONGENITAL H.D. - WHEN FIRST NOTED _____

_____

HOW LONG IS CHILD KNOWN TO HAVE HEART DISEASE_____

WHEN AND BY WHOM NOTED _____

RHEUMATISM - DATE FIRST ATTACK_____ SUBSEQUENT ATTACKS_____

PHYSICAL EXAMINATION ON ENTRANCE TO SPALDING SCHOOL

DATE:_____
GENERAL CONDITION
THROAT_____ TEETH_____ TONSILS_____ EARS___ NOSE____

HEART:
RATE APEX_____ RADIAL_____
RHYTHM_____ APEX BEAT_____
THRILL_____ SIZE _____
SOUNDS_____ MURMURS_____
BLOOD PRESSURE_____ URINALYSIS_____
EKG_____ X-RAY

CARDIAC DIAGNOSIS _____

_____

CLASSIFICATION_____ OTHER LIMITING DISABILITIES_____

SPECIAL NOTES_____

_____

_____

PROGRESS NOTES _____

_____

Fig. 3.—Medical history and results of physical examination on entrance to school, as well as subsequent observations.

The diagnoses on entrance are tabulated in Table II. Eighty-five per cent had rheumatic heart disease and 9.4 per cent had congenital heart disease. It should be noted that these figures are not comparable to those obtained in studies on large groups of children in public schools, where the incidence of rheumatic heart disease is stated to be 55 per cent,[7] 65 per cent,[8] or 69 per cent[9] of all organic heart disease found in school children. White and Jones's study, in 1928 (referred to by White[10]), showed that rheumatic heart disease constituted 92 per cent, and congenital heart disease, 6 per cent, of all children's heart

disease. The slight discrepancy here can be explained by the fact that physicians seem more likely to seek admission for those children with congenital heart disease, especially when they are cyanotic, than for those with rheumatic heart disease in Class I or IIA. The physician is often willing to allow the parents of children with rheumatic heart disease of Class I or IIA to send them to a regular school. The majority of admissions were in cardiac classification IIA or IIB (Table III). Patients placed in Class III were automatically denied admission, but were allowed to reapply upon improvement.

HOME FOLLOW-UP #4
FAMILY

NAME	KIN	AGE	HEALTH	OCCUPATION	INCOME

HOME:
TYPE OF BUILDING _____ LOCATION OF BUILDING _____
NO. OF ROOMS _____ HOW HEATED _____
RENT OR OWN _____ NEIGHBORHOOD _____

PATIENT

H.S. - SPECIAL _____ COURSE _____ GRADES _____
REGULAR _____ _____ _____

YRS. IN EL. SCHOOL _____ YRS. IN H.S. _____
EXTRA CURRICULAR WORK
WHILE IN H.S. _____
CAMP EXPERIENCE - WHERE _____ WHEN _____
RECREATION _____
MARRIED _____ CHILDREN _____
TYPE OF WORK DONE SINCE LEAVING SCHOOL _____ HOW LONG _____ WAGES _____

PRESENT JOB, IF ANY _____

APPROX. INC. EARNED PAST YR. _____ TRANSPORTATION USED _____

GENERAL HEALTH
ANY ATTACKS OR HOSPITALIZATIONS SINCE GRADUATION, WHEN & WHY _____

	DATE	IMPROVED	WORSE	SAME
MOST RECENT PHYS. EX.				

CLINIC AND NUMBER _____
or
PRIVATE PHYSICIAN _____

COMMENTS _____

Fig. 4.—Current social and economic data secured by home call.

The average age at entrance was 12 years. Metropolitan Life Insurance Company[11] figures give the age of onset of rheumatic fever at "from 5-9," and the average age of onset of rheumatic heart disease as "after 10." White[10] allows a larger latitude: "between the ages of 4

TABLE II

CARDIAC DIAGNOSIS UPON ENTRANCE TO SPALDING SCHOOL

DIAGNOSIS	CASES WITH FULLY COMPLETED RECORDS	DIED SINCE LEAVING SCHOOL	OUT OF TOWN	UNABLE TO LOCATE	NO COOPERATION	TOTAL
Rheumatic heart disease	116	43	17	15	7	198
Congenital heart disease	10	10	2	1		23
Following diphtheria	1					1
Paroxysmal tachycardia	1					1
Renal hypertension	1					1
Congenital with superimposed rheumatic heart disease	1					1
Miscellaneous		3	2	3		8
						233

PHYSICAL EXAMINATION

DATE_____

NAME_____AGE_____S M W D SEP._____

ADDRESS_____

DAYS LOST DUE TO ILLNESS
WHILE AT WORK - HOME_____CON. HOME_____HOSP._____

NATURE OF ILLNESS_____

NEW SYMPTOMS_____

PHYSICAL EXAMINATION:
WEIGHT_____HEIGHT_____TEMP._____RESP._____

GENERAL_____

HEART:
RATE_____

APEX_____

RADIAL_____

RHYTHM_____

APEX BEAT_____

THRILL_____

SIZE_____

SOUNDS_____

MURMURS_____

BLOOD PRESSURE_____

EKG_____X-RAY_____

CARDIAC DIAGNOSIS_____

_____

_____CLASSIFICATION_____

LIMITING DISABILITIES AS THEY WOULD AFFECT EMPLOYMENT_____

_____

_____

PHYSICIAN_____

Fig. 5.—Recheck physical examination done at the office of the Chicago Heart Association by Dr. Arthur F. Abt or Dr. Kate H. Kohn.

TABLE III

CARDIAC CLASSIFICATION ON ENTRANCE TO SPALDING SCHOOL

CLASSIFICATION	CASES WITH FULLY COMPLETED RECORDS	DIED SINCE LEAVING SCHOOL	OUT OF TOWN	UNABLE TO LOCATE	NO COOPERATION	TOTAL
E	2			1		3
F	4		1	3		8
I	5	1	3	1		10
IIA	63	20	9	9	6	107
IIB	56	35	8	5	1	105
						233

TABLE IV

NATIONALITY OF FAMILIES

NATIONALITY	CASES WITH FULLY COMPLETED RECORDS	DIED SINCE LEAVING SCHOOL	OUT OF TOWN	UNABLE TO LOCATE	NO COOPERATION	TOTAL
American	31	12	9	9	3	64
Assyrian		1				1
Bohemian	5					5
Canadian	1					1
Czechoslovakian	3					3
Danish	1					1
English	2	1				3
German	4	4	1	1		10
Greek	3	1				4
Irish	9	2	2			13
Italian	18	9		1	2	30
Lithuanian	2	2	1			5
Norwegian		1				1
Polish	13	5	2	5	1	26
Russian	17	12	2	2		33
Scotch	2					2
Swedish	1	2				3
Ukranian	1					1
Bohemian-Irish	1					1
Croatian-Hungarian	1					1
Dutch-German	1					1
English-Irish	1					1
French-Austrian	1					1
German-Scotch	1					1
Irish-German	2					2
Italian-German	1					1
Polish-Hungarian	1		1			2
Polish-Lithuanian	2					2
Russian-Lithuanian	1					1
Scotch-Irish	4					4
Austrian-Hungarian		1				1
German-American					1	1
German-Hungarian		1				1
German-Polish		1	1			2
Russian-German		1				1
Irish-Hungarian			1			1
French-American			1			1
Irish-American				1		1
						233

and 15 years, the height of onset (of rheumatic heart disease) being between the seventh and eighth years."

The nationality of the families of the students was as varied as would be expected in a city like Chicago. Americans made up the largest group, and Russians, Italians, and Poles followed in that order (Table IV). No racial or national group has ever been found to be resistant to either rheumatic or congenital heart disease.[9, 10] Only seven of our patients were Negroes, but this was due to the location of the school in the city, and not to a resistance on the part of that race.

Most of the families had four or five children, although one fairly large group of those who died after leaving school were from families with six or more children. The families were rated by the nurse when she made her home calls as either good, fair, or poor from a socioeconomic point of view. Thirty-eight per cent were rated good; 39 per cent, fair; and 23 per cent, poor. These groupings must be compared to the ratings of a public school group in a big city, and not to the population at large, for rheumatic fever is known to be one hundred times more common in the poorer socioeconomic strata. Rheumatic fever and rheumatic heart disease are rarely found in private schools.[10] Also, the Spalding School is located in an exceedingly "poor"[9] neighborhood, and the best of its families are in the lower income brackets.

Our patients were approximately equally divided as to sex, and no difference in severity of the heart disease in either sex was noted.

The grade placement of children with heart disease is affected by their condition only insofar as it keeps them out of school for long periods of time. Otherwise, our students were in the grade in school which was commensurate with their age and mentality, as is the well child. The school itself is able to meet the educational requirements of a standard Illinois elementary and high school.

Thirty-five of these students had been given a psychiatric interview.* Seventeen of these are living, and eighteen died after leaving school. Two-thirds of the group appeared to recognize their handicap and to accept it for the most part. Their acceptance had a definite correlation with their identification with the school and their enhanced feeling of security while in school. Some of those who felt or showed this increased security stated that it was because they were one of a group, and not one person singled out for special emphasis because of heart disease. Also, many said they knew being at the school would help their condition, and so eagerly accepted it. Lyon, et al.,[12] whose group of twenty-two children with heart disease were attending regular school, found that many of the special problems of these children, such as worry over, or resentment against, his illness, concealment of it in order to increase his activity, etc., were removed by "conferences with school teachers and school principals, by advice to parents and other children in the family and by the provision of special activity and vocational

*Conducted by Dr. Paul Schroeder and associates at the Institute for Juvenile Research in Chicago.

training for the patients." In general, the interviews led us to agree with Silver:[13] "That the presence of these handicaps has not caused any marked deviations from the standards of ordinary conduct or any disturbance in the development of normal social relationships."

The average length of stay in Spalding School was 2.4 years, although some students attended other special schools in the city before or after Spalding School, and so presumably were in a special school longer. Many patients, if improved while in Spalding School, are labeled "no further special school care felt to be necessary" by their own physician or the Chicago Heart Association school physician. Various other reasons for leaving Spalding School are shown in Table V. It is interesting that only seven out of 233 left against medical advice, which means that no possible excuse for considering them improved enough to be classified in the first group could be found. Three of the seven later died. Only nine out of a total of 109 considered improved enough to leave were in the died-since-leaving-school group. It should be further noted that 19 out of 26 who left school because of the severity of their illness later died.

TABLE V

REASONS FOR LEAVING SPALDING SCHOOL

REASON FOR LEAVING	CASES WITH FULLY COMPLETED RECORDS	DIED SINCE LEAVING SCHOOL	OUT OF TOWN	UNABLE TO LOCATE	NO CO-OPERATION	TOTAL
No further special care felt necessary	76	9	11	12	1	109
Graduated, high school	15	10			1	26
Moved	5	5	6	3	1	20
Working	12	5	2	1	2	22
Overage (staying at home)	9	4	1	1	1	16
Ill at home	3	15	1	1		20
Ill, hospitalized	2	4				6
To parochial school	2			1		3
Left against medical advice	3	3			1	7
Transferred—distance to special school too great	1	1				2
Dismissed to Municipal Tuberculosis Sanitarium (pulmonary tuberculosis)	1					1
Dismissed—pregnant	1					1
						233

The cardiac diagnosis and classification on leaving the school remained the same as that made on entrance in most instances. A few children were thought to have developed further valvular damage, but, in the great majority, clinical observation showed no progression of disease. Occasionally, variations in the clinical signs in cases of congenital heart disease would change the anatomic diagnosis, but it is known that the clinical signs in congenital heart disease are often in-

constant, and therefore this does not imply that the patient's condition is worse.

As previously stated, 130 patients were interviewed and examined to ascertain what had happened to them since leaving the school. The youngest was 17 years old, and the oldest, 33, at the time of the recheck physical examination. There were 65 males and 65 females. Thirty-two of the women were married: fourteen had borne one child; three had two children; one had three children; two had had four pregnancies; three were pregnant. None reported that her heart condition had adversely influenced her pregnancy or labor to any great extent. All of these women stated that the medical care given them during their pregnancies and labors took into account their heart condition. Martin[14] reports similar observations. Stroud and Twaddle[15] reported that one out of 27 had congestive failure at the time of delivery.

One hundred nineteen (60 males, 59 females) reported that they either saw a physician regularly or had been examined at least within the previous three years. Seven (2 males, 5 females) had not seen a physician within three years, but had done so prior to that time. Four (3 males, 1 female) had had no medical care since leaving school. Presumably the effort made in school to educate them to the necessity of frequent medical checkups had some effect. It should be further noted that there were few intervening illnesses, operations, or accidents which required medical attention, other than that necessitated by their cardiac condition.

Twenty-six (20 per cent) had recurrences of rheumatic fever: 13 had one recurrence, 10 had two, one had three, one had five, and one had ten (the latter patient, incidentally, is now quite well and in cardiac classification IIA). Wilson[16] states that the number of recurrences of rheumatic fever tends to decrease after puberty. According to Cohn and Lingg,[17] there is "diminished susceptibility to rheumatic infection at puberty." Therefore, this low percentage of recurrences was to be expected, for most of these students were approaching puberty, or were beyond it, when they left school.

The diagnoses, on the whole, remained the same as those made on leaving school. Because of the lapse of time since the initial infection, it was possible to discard a diagnosis of "potential" or "possible" heart disease for a definite diagnosis of "no cardiac disease." In five cases "auricular fibrillation" had to be added to the diagnosis, and one patient had had auricular fibrillation for two years, but not at the time of the recheck examination. This patient had had five recurrent attacks of rheumatic fever. Of the other patients who had auricular fibrillation, three had had two recurrent attacks of rheumatic fever and two had had one recurrent attack each. Four were classified in Class III and two in Class IIB. Table VI lists fourteen cases of auricular fibrillation, with the number of years during which it is definitely known to have been present. Four cases are from this study. Ten

TABLE VI

CASES OF AURICULAR FIBRILLATION

NUMBER	CODE NUMBER	SEX	AGE AT RECHECK EXAMINATION	ENTRANCE DIAGNOSIS	KNOWN DURATION OF FIBRILLATION (YR.)	COMMENT
1		F	20	Rh. M. I. and S.	4	Completed high school in 4 years without remission of fibrillation
2	17*	F	19	Rh. M. I. and S.	1	
3		F	19	Rh. M. I. and S.	4	Completed high school in 4 years
4		M	22	Rh. M. I. and S. A. I. and S.	1	Completed high school in 5 years with fibrillation during last year
5		M	16	Rh. M. I. and S.	4	
6	13*	F	21	Rh. M. I. and S. A. I. and S.	2	Five recurrent attacks of rheumatic fever
7	161*	M	26	Rh. M. I. and S.	8 mo.	Two recurrent attacks of rheumatic fever
8	27*	F	24	Rh. M. I. and S.	4	One recurrent attack of rheumatic fever
9		F	21	Rh. M. I. and S.	4	
10		M	*Age at Death* 19	Rh. M. I. and S. A. I. and S.	2½	Fibrillated continuously last 2½ years of high school. Completed high school in 4 years. President of his class
11		M	24	Rh. M. I. and S. following scarlet fever	3	Completed high school in 4 years without remission of fibrillation
12		M	18	Rh. M. I. and S.	6	Four years high school with demise 2 months before graduation. Ranked first in his class. Worked after school last 2 years
13		F	17	Rh. M. I. and S.	1	
14		M	15	Rh. M. I. and S.	1	

*Refers to case number in Table I.

others—students at Spalding School, but outside the scope of this study—are included because of interest in the clinical condition and general picture of children who have auricular fibrillation over a long period of time, rather than as a terminal event. Nine (3 males, 6 females) are living. The youngest was 16, the oldest, 26, years of age. The duration of auricular fibrillation varied from eight months to four years; four of these patients have had it for four years. Five (4 males, 1 female) of them are dead. The youngest was 15, the oldest, 24, years of age. Two had had auricular fibrillation one year before death, one, two and one-half years, one, three years, and one, six years, so that

their period of fibrillation could hardly be considered a terminal event. The great majority of them knew when they had auricular fibrillation, but did not exhibit any undue concern over their heart condition. In general, they did well in school.

It is interesting to compare the cardiac classification on recheck examination with that on leaving school. The number of years since these students left the school varies from one to 18 years. Of the 76 who left school because they were considered well enough to attend regular school (see Table V), 24 (6, IIB; 15, IIA; 3, I) are better, 36 (5, F or E; 8, I; 18, IIA; 5, IIB) are the same, 16 (2,F; 3, I; 9, IIA; 2, IIB) are worse, and 2, classed as IIB, are now Class III.

Of the 15 who left because they had completed high school, 6 (1, E; 2, IIA; 3, IIB) are better, 6 (3, IIA; 3, 11B) are the same, 3 (2, IIA; 1, IIB) are worse, and the last-mentioned IIB is in Class III. Of the 5 who moved out of the district, 3 (1, IIA; 2, IIB) are better, one IIB is the same, and one IIA became a Class III.

Of those who left to go to work, 6 (2, IIA; 4, IIB) are better, 5 (1, I; 3, IIA; 1, IIB) are the same, and one IIA became IIB. Of those who stayed at home, 2 in Class IIA are better, 5 (3, IIA; 2, IIB) are the same, 2 (1, I; 1, IIB) are worse, and the last-mentioned IIB is in Class III. Of those too ill to attend school, 2 (1, IIA; 1, IIB) are the same, and one in Class IIA is now in Class IIB.

Of those hospitalized, two in Class IIB are better. Two in Class IIA left to go to parochial school, and are now in Class IIB. Three left against medical advice: two in Class IIB are better, and one in Class IIA is the same. One in Class IIB who lived too far away to continue at the school is now in Class I. One in Class IIB with active pulmonary tuberculosis is now in Class IIA, but is a schizophrenic. One in Class IIA who left because of her pregnancy is now in Class I. To summarize: 48 are better, 56 remained the same, and 26 are worse. Of the five in Class III, all have auricular fibrillation.

It is interesting to note what these persons are able to do. Almost all of them are working. The jobs held cover a wide field:

Commercial art work:
- Poster artist
- Greeting card artist
- Plaster art worker
- Sign painter

Dental technician
Librarian
Photographer
Photographer's model
Piano teacher
Reporter (newspaper)
Soldier (U. S. Army)
Student
Office work:
- File clerk

Window decorator
Factory:
- Candy packer
- Package wrapper
- Packer
- Punch press operator
- Receiving room (clerk)
- Shipping clerk

Barber
Butcher
Carpenter's helper
Electrician's helper
Machinist helper
Plasterer's helper
Plumber's helper

- Stenographer
- Switchboard operator
- Typist

Retail business:
- Purchasing agent
- Order filler
- Shoe store owner

Cashier

Clerk:
- Department store
- Bakery
- Grocery

Dressmaker

Solicitor (outside work)

Theatre usher

"26" girl

Printer's helper

Tool die maker

Cab driver

Car washer

Coal man

Delivery and errand:
- Pharmacy
- Florist
- Stock broker

Domestic:
- Hotel
- Private family

Repair man (hardware store)

Subway mucker

Truck driver

Waiter

Waitress

Only five had never worked or were not working at the time of this study. Many were doing heavy work. Fifty per cent of the women were doing their own housework and taking care of their children as well, and let no one say this is not heavy labor. One man had been accepted by the Army and was in active service. One traveling salesman carried his heavy sample suitcases all day, and climbed many flights of stairs with them.

Their recreational activities cover just as wide a field as did their jobs:

Home

Art work:
- Charcoal drawing
- Water coloring
- Clay modeling

Beauty culture

Cards

Cooking

Crocheting

Knitting

Mechanics

Printing

Photography

Philately

Radio

Reading

Sewing

Music:
- Accordion
- Clarinet
- Piano
- Violin
- Singing

Community activities:
- Boy Scouts
- Girl Scouts
- Church socials
- Clubs
- Dances
- Movies

National Guard

Out of doors and sports:
- Baseball
- Basketball
- Bathing
- Bicycling
- Bowling
- Fishing
- Football
- Golf
- Gymnastics
- Hiking
- Horseback riding
- Hunting
- Automobile rides
- Roller skating
- Skiing
- Speed boating
- Swimming
- Tennis
- Travel

These are recreations common to all young people, and the cardiac condition seems to have exerted little influence except insofar as the person was actually limited to sedentary activity. Dancing was a favorite of many, and can scarcely be considered sedentary.

We have been unable to locate 19 of the students. Seven refused to come for examination or to answer any questions, and 21 were reported to be living out of town. However, these latter groups should be considered among the living. Fifty-six are known to be dead, making a mortality of 26 per cent for the entire group. Stroud and Twaddle[15] reported 21 per cent mortality in their series, and quoted Jones's report of 24.2 per cent mortality in his series and Halsey's report on Irvington House patients as 24 per cent. Martin[14] reports 29.9 per cent mortality in his group. However, it must be noted that ten of the 56 who died had congenital heart disease—a total of 18 per cent. Of the group of 158 known to be alive, only 12, or 7.6 per cent, had congenital heart disease. Hedley[18] states that most observers consider "approximately 10 per cent" of deaths from heart disease among young persons to be of congenital origin. Our figure of 18 per cent is higher because this was a selected group of children with severe heart disease who were well enough to attempt some school activity. Children of school age with severe congenital heart disease are more likely to be sent to a school of this kind, if it is at all possible, than children with severe rheumatic heart disease.

The length of time these children lived after leaving the school varied from one to eleven years. Of 9 who left because no further special school care was considered necessary (see Table V), 2 lived eleven years, 1 lived nine years, 1 lived eight years, 1 lived six years, 1 lived four years, 1 lived three years, 1 lived two years, and 1 lived one year. But of those 19 who left because they were too ill to remain in school, 1 lived eleven years, 1 lived nine years, 1 lived five years, 1 lived three years, 5 lived two years, and 10 lived one year or less. Three left against our advice—2 of them had congenital heart disease. One lived two years, 1 lived four years, and 1 lived three years after leaving school. It is interesting that 41 out of this group of 56 had done no work of any kind—even light housework—since leaving school. Considering their economic level and the fact that all the others mentioned some job, however small, it is presumable that these 41 were too ill to work.

We were able to obtain forty-three death certificate reports. Twenty-four definitely stated or clearly implied that the "cause of death" (or one of the contributing factors) was rheumatic heart disease. In eleven cases in which the same diagnosis had been made during life, the "cause of death" was "chronic myocarditis" or an equally nonspecific diagnosis. In eight cases the ante-mortem diagnosis was congenital heart disease. According to the death certificates, two of these patients had died of rheumatic heart disease, one of chronic myocarditis, one of bacterial endocarditis with congenital heart disease as a contributing

factor, one of malignant endocarditis (with no mention of the congenital deformity, even though an autopsy was performed), one of streptococcic meningitis with congenital heart disease a contributing factor, one of morbus caeruleus and patent foramen ovale, and one of cerebral embolism and endocarditis. Two patients died of cerebral embolism. One of them had subacute bacterial endocarditis, and both conditions were found at autopsy; the other had a diagnosis, before death, of congenital heart disease with superimposed rheumatic heart disease. There were three who died of subacute bacterial endocarditis; all three had had rheumatic heart disease. One patient died of lobar pneumonia; his rheumatic heart disease was not mentioned on the certificate. Only four autopsies had been performed; two confirmed diagnoses of rheumatic heart disease; one, previously mentioned, confirmed the diagnosis of cerebral embolus and subacute endocarditis; one, also previously mentioned, was reported as confirming the diagnosis of the cause of death as malignant endocarditis, without any mention of the congenital deformity that was known to have been present.

COMMENT

This is a survey of a group of children with heart disease whose medical treatment was supplemented by their attendance at a special school. Evidence is here presented that these children progressed favorably from the clinical point of view. In addition, they lived at home and continued their education in a normal manner. They and their families received continual intensive advice and guidance through the normal ordinary channels of parent-teacher-school relationship. It should be further noted that patients with congenital heart disease with marked cyanosis—usually severely handicapped—were also enabled to attend school without embarrassment or risk. Authorities[2, 14, 15] agree that patients who have had rheumatic fever need a long convalescence, either to prevent the development of heart disease or to treat the heart disease which has already appeared, and that this is best done in a convalescent home. In his survey of facilities available for the care of cardiac children, Hedley[19] discusses types of convalescent institutions in detail, and states that treatment should be planned for "about one year." Of the twelve states who had cardiac programs in operation by January, 1942, eleven had made provision for sanitarium care, and most of them had also provided some plan of foster home care.[20]

A typical state program, as outlined by Galvin,[21] attempts to provide treatment for the sick child and follow-up care for the convalescent child, but it makes no provision for control of the child over a long period of time. However, Galvin[21] states: "information regarding the school child is sent promptly to the school principal and school nurse to protect him not only from undue exposure to colds and unwise activities but also from oversolicitude." In a special school, the child remains in his own home, yet receives such protected schooling. Also, both he and his parents are educated to the special needs of heart dis-

ease in childhood and in the future. Much parental anxiety is removed, and this tends to relieve the cardiac child of one of his greatest handicaps—the oversolicitous and fearful parent.[13, 22]

Utter[23] stresses the need of informing the child about his own care and the avoidance of future infection. Siegel[24] states that children with congenital heart disease whose lesions, although severe, are compatible with a life of limited activity should have "regulation of exercise, anticipation and prevention of intercurrent disease and education for a suitable vocation." White[25] and his group conclude that the child's cooperation is best when the environment is made as homelike as possible. Martin[14] mentions the importance of avoiding wrong diagnoses in order to prevent cardiac neuroses. Also, he stresses the avoidance of partial invalidism among those with heart disease who are able to lead normal, if limited, lives.

In general, authorities are agreed on the need for care of children with rheumatic heart disease long after the initial infection. Convalescent homes are best for that desirable "continuity of care"[26] immediately after the initial illness or the recurrence. But, for long periods thereafter, patients with inactive rheumatic heart disease and children with congenital heart disease need limited activity, medical supervision, and guidance and education for the future. Obviously, as Galvin[27] points out, only limited numbers of patients can be cared for in institutions and foster homes. The special school permits shortening of the period of time during which these facilities are required. Special facilities, such as those described for Chicago, in the local public school system offer any cardiac child the opportunity for a school life which is made more normal by the fact that he is enabled to live with his own family in his own home and to attend school as other children do.

SUMMARY

1. The importance of the public health aspect of cardiac disease in children is discussed briefly.

2. A school program for cardiac children is described.

3. Data are presented on a follow-up survey of all students who had attended such a school. This covered approximately a ten-year period. Of the total of 233 students, 158 were living and 56 were dead.

4. Of 130 examined, 104 had either improved or remained in the same cardiac classification since leaving the school. Their current activities are described.

5. A special school for cardiac children serves the following purposes:

a. It can carry on where the convalescent home leaves off, and can continue for much longer periods of time the kind of medical supervision a child with rheumatic heart disease requires.

b. A more normal life for cardiac children of school age with limited capacity for any activity is provided.

c. Contact is made with the parent, as well as the child, and thus

both can be educated in the practical aspects of the type of living suitable for cardiac children and adults.

Dr. Arthur F. Abt, formerly chief Chicago Heart Association physician at the Spalding School, and now on military leave of absence, initiated this study and has given much of his time and advice to its execution.

We wish to thank Dr. Isaac A. Abt, Miss Elizabeth Stern, and Miss Grace Bennett for their assistance.

The school records were made available to us by the Board of Education of the City of Chicago.

Death certificates were made available to us by the Bureau of Vital Statistics, Cook County Clerk's Office, City of Chicago.

School nurses stationed at the Spalding School by the Board of Health of the City of Chicago assisted in the recheck physical examinations.

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Clinical Reports

ELECTROCARDIOGRAPHIC CHANGES SIMULATING THOSE OF ACUTE MYOCARDIAL INFARCTION IN A CASE OF PERFORATED GASTRIC ULCER

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THE differential diagnosis between myocardial infarction and acute surgical lesions of the upper abdomen is at times difficult, yet obviously of the greatest importance, for the former demands conservative management, whereas the latter may require immediate surgical intervention.

The fairly characteristic electrocardiographic patterns which occur with myocardial infarction make the electrocardiogram a valuable adjunct in the differentiation of these two types of acute disease. However, to interpret properly the electrocardiographic data obtained under these circumstances, certain pitfalls must be avoided, one of which is the not infrequent absence of electrocardiographic changes early in myocardial infarction. Second, one must bear in mind the occurrence of single or multiple coronary artery occlusions in elderly patients in shock,[1] a situation in which myocardial infarction occurs in association with, and probably as an indirect result of, the acute surgical or "shocking" disease. The case described here represents still another pitfall in diagnosis, namely, electrocardiographic changes suggesting acute myocardial infarction without coronary artery occlusion, but in the presence of a ruptured abdominal viscus with associated shock.

REPORT OF CASE

H. V., a white man, aged 68 years, was admitted to the Hospital of the University of Pennsylvania Feb. 8, 1943. On the day of admission, while en route to the Gastrointestinal Clinic, he became weak, collapsed on the street, and was brought into the Hospital by the police. His history was one of upper abdominal pain for the preceding ten years. The pain was dull, nonradiating, occurred usually ten to fifteen minutes after meals and during the night, and was relieved by alkali. During the two months prior to admission, he had lost about 40 pounds in weight, probably because of marked anorexia. During this time he also had melena, hematemesis, and mental confusion, and had resorted to the use of sodium bicarbonate every few hours. There was no history of exertional dyspnea or angina of effort.

Physical examination on admission revealed an emaciated white man who appeared anemic, dehydrated, and chronically ill. The tempera-

From the Medical Clinic and the Edward B. Robinette Foundation, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.
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ture was 98° F., the pulse rate, 78, and the blood pressure, 170/100. The chest was emphysematous. The heart did not seem enlarged on clinical examination. The first heart sound was distant, the aortic second sound was accentuated, and a soft blowing systolic murmur was heard at the apex. Examination of the abdomen revealed moderate distention and slight bilateral upper abdominal tenderness without rigidity. The edge of the liver was felt 2 cm. below the right costal margin, and was not tender. Deep palpation just below the liver margin revealed a somewhat tender, round, ill-defined mass, apparently about 6 cm. in diameter, which did not move on respiration. The peristaltic sounds were of normal frequency and pitch. Rectal examination was noncontributory. Examination of the extremities revealed obvious and marked peripheral arteriosclerosis. The tentative diagnosis on admission was chronic peptic ulcer, probably with malignant change.

Laboratory studies disclosed an erythrocyte count of 4,200,000, a hemoglobin of 11 Gm. (75 per cent), and a leucocyte count of 9,400. The neutrophiles were 66 per cent, the lymphocytes, 24 per cent, the monocytes, 9 per cent, and the eosinophiles, 1 per cent. Urinalysis showed a specific gravity of 1.022, 1 plus albumin, no sugar, and only an occasional leucocyte per high-power field. The serum CO_2 was 94 volumes per cent, the serum chloride, 83.9 meq. per liter, and the serum proteins, 6 Gm. per 100 milliliters.

The patient was allowed nothing by mouth, and an intravenous infusion of 5 per cent glucose in .85 per cent sodium chloride solution was given. By the morning of Feb. 9, 1943, he was feeling much improved. His temperature was 98.3° F. The hemoglobin had fallen to 48 per cent and the leucocyte count to 6,000, probably as a result of better hydration. Frequent bland feedings were begun.

At noon on Feb. 10, 1943, the patient suddenly developed severe epigastric pain, with tenderness and rigidity of the entire abdomen and absence of peristalsis. The blood pressure fell to 60/30, the pulse rate rose to 125, and the patient appeared to be in extreme shock. A diagnosis of ruptured peptic ulcer was made, with which a surgical consultant agreed. However, an electrocardiogram taken at this time (Fig. 1) revealed S-T segment depression in Leads I, II, CR_4, and CR_5, strongly suggesting infarction of the lateral wall of the left ventricle. Nevertheless, in spite of this, the history and physical examination seemed to point clearly toward an abdominal catastrophe, perhaps associated with acute myocardial infarction. The surgical consultant felt that the almost moribund condition of the patient contraindicated immediate operation, and the usual measures to combat shock were taken. The temperature rose to 101° F. (rectal), the blood pressure remained quite low, the pulse became imperceptible, and the patient died seven hours after the onset of the collapse.

NECROPSY

The necropsy, performed by Dr. Melvin Friedman on Feb. 11, 1943, revealed the following: There was generalized peritonitis, and culture of the peritoneal exudate showed nonhemolytic streptococci, *Streptococcus fecalis*, and *Bacillus coli*. The lungs were congested. The spleen and kidneys showed moderate arteriolarsclerosis. The pancreas, liver, gall bladder, adrenals, and prostate were normal. There was an extensive ulcer crater about 6 cm. in diameter and 1 cm. in depth in the prepyloric region of the stomach on the lesser curvature. On the anterior side of the ulcer there was a 1 cm. perforation. Microscopic examination revealed no evidence of malignant change. The heart, which

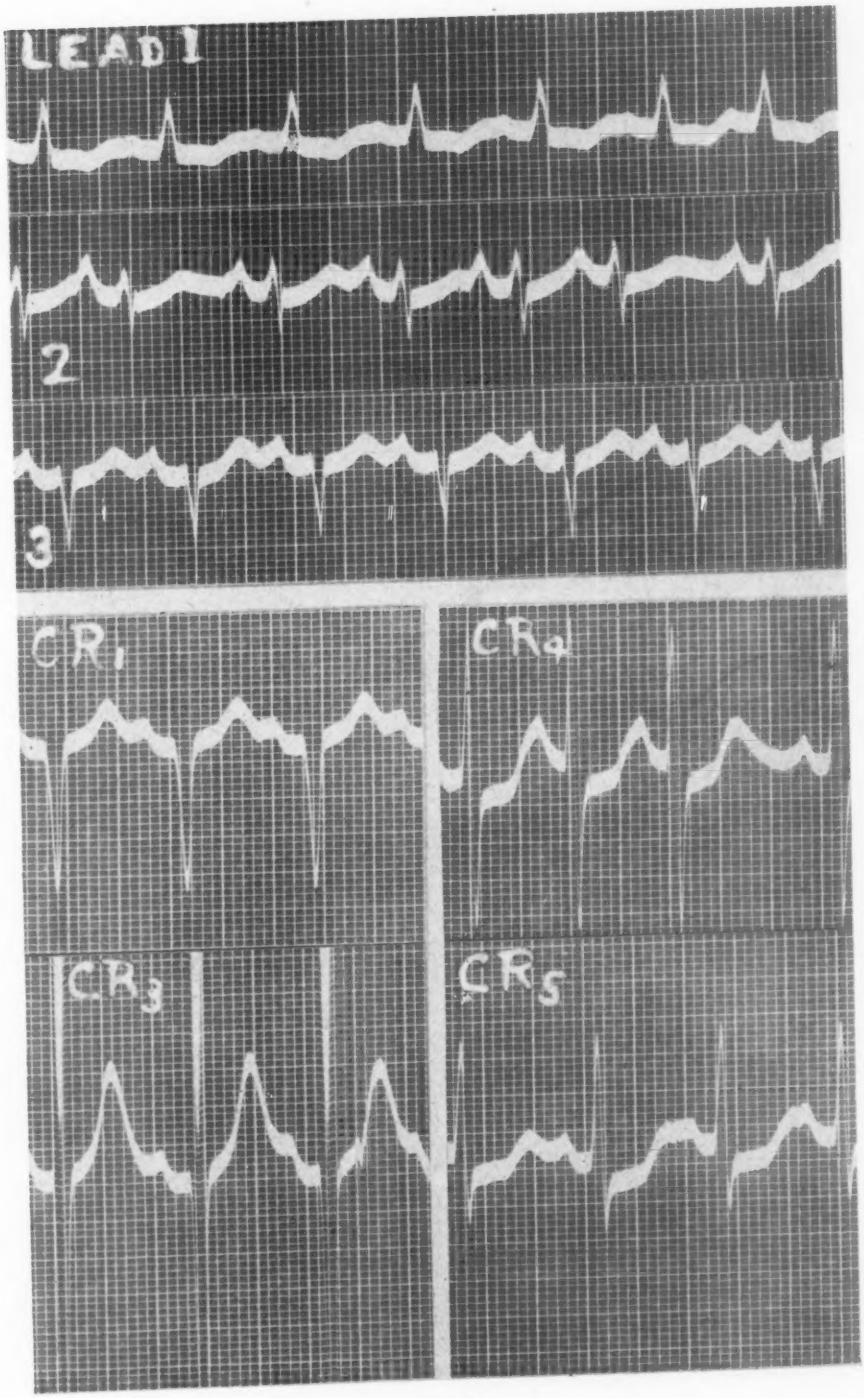

Fig. 1.

weighed 430 grams, was examined with the greatest care by Dr. Friedman and one of us (F.D.M.). The coronary vessels were followed out to their smallest radicles. The arteries were quite sclerotic, particularly the circumflex branch of the left coronary. However, no fresh or old occlusions could be demonstrated. The heart was next hardened in formalin for several days, then cross-sectioned in slices 1 cm. thick from apex to base. In scattered areas of the myocardium, with no distinct anatomic distribution, there were a few tiny spots of fibrosis. Histologic study of multiple areas of the myocardium, particularly the lateral wall of the left ventricle, revealed no abnormalities except occasional small areas of old fibrosis.

DISCUSSION

That abdominal lesions may simulate coronary occlusion closely has been clearly recognized, and the diagnostic difficulties resulting therefrom are well illustrated, for example, by the reports of Barker, et al.,[2] and Averbuck.[3] The story of the acute attack and the physical examination alone may be confusing, but, when accompanied by a long-standing history of mild to severe cardiac distress, the diagnostic problem often becomes highly perplexing. Since the electrocardiogram would appear to be a logical aid in differential diagnosis, it becomes important then to recognize its limitations under these conditions.

Fitz-Hugh and Wolferth[4] have demonstrated electrocardiographic changes, mainly flat or inverted T waves in Leads I and II, which occurred in the presence of gall bladder disease and disappeared after appropriate surgical intervention. These changes they interpreted as meaning that gall bladder disease in some as yet unexplained fashion affects the myocardium. McGee, et al.,[5] have reported a case of perforated duodenal ulcer with small but definite S-T segment elevation in Leads II, III, and a chest lead, as well as T-wave inversion in Leads II and III. This patient had a history of exertional epigastric pain, making the diagnosis yet more difficult. Necropsy showed little evidence of coronary artery disease. These authors felt that insufficient oxygenation of the myocardium resulting from the low blood pressure of shock was the probable explanation of the electrocardiographic changes. Gottesman, et al.,[6] have reported a case of acute pancreatitis with depressed S-T segment in Leads II and III and a chest lead, and diphasic T waves in Leads I, II, and III, and a chest lead. Upon recovery, the patient had a quite normal electrocardiogram.

In addition to this electrocardiographic mimicry of myocardial infarction, one must also recognize the possibility of the actual coexistence of abdominal disease and coronary occlusion. Blumgart, et al.,[1] have emphasized the occurrence of multiple coronary artery thrombi in patients with antecedent shock. The majority of their patients were elderly, were known to have had pre-existing heart disease, and at necropsy showed evidence of coronary artery disease. No electrocardiographic data were given, however.

The explanation for the abnormal electrocardiographic patterns in our case can be only speculative. Unfortunately, no previous electrocardio-

grams had been taken. Actual arterial occlusion seems to have been ruled out by careful post-mortem study. The theory that the changes were "reflex" in origin would seem to have little clinical or experimental evidence in its favor. Since the systemic blood pressure at the time the electrocardiogram was taken had fallen to 60/30, and the coronary arteries were shown to have greatly narrowed lumina, it would seem more logical to attribute the abnormal patterns to impaired blood supply to the myocardium, with resultant poor oxygenation of the muscle. In fact, the electrocardiographic changes in this case are quite similar to those which occur during an anginal attack or during the breathing of an oxygen-poor mixture.[7] It seems clear, therefore, that, in a case of coronary arteriosclerosis, the possibility exists that any disease causing shock may give rise to electrocardiographic patterns of the "anoxemic" type, which, since they are so much like those seen in lateral wall infarction, may lead to an erroneous diagnosis of coronary occlusion. This likelihood of error in diagnosis becomes still greater when the clinical observations are equivocal, a situation often present with acute surgical lesions of the upper abdomen.

SUMMARY

A report is presented of a case of perforated gastric ulcer, with shock, in which an electrocardiogram strongly suggested infarction of the lateral wall of the left ventricle. Careful post-mortem study of the heart revealed arteriosclerotic narrowing of the coronary vessels, but no evidence of actual occlusion or of myocardial infarction.

The presence of marked coronary artery narrowing and the fact that the blood pressure was greatly lowered have led us to postulate insufficient blood supply to the myocardium and, therefore, anoxemia of the muscle as the cause of the electrocardiographic abnormalities.

It is emphasized that the electrocardiogram can play an important role in the differential diagnosis between coronary artery occlusion and acute abdominal lesions, but that it may be confusing and actually misleading unless one recognizes its limitations and possible mimicry under these conditions, particularly when shock is present.

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Abstracts and Reviews

Selected Abstracts

Nickerson, J. L., and Curtis, H. J.: The Design of the Ballistocardiograph. Am. J. Physiol. **142:** 1, 1944.

An experimental and theoretical study of the human ballistocardiograph has been undertaken with a view to designing an apparatus which will measure accurately and easily the motion of the body which occurs as a result of the heartbeat. Both approaches to the problem have demonstrated that:

It is absolutely necessary to have the bed damped by a device which introduces no friction. Since both over and under damping yield errors which are difficult to evaluate, the damping should be critical for the total load.

The restoring force on the bed is most easily specified by the undamped frequency of oscillation of the loaded bed. True ballistic records are obtained only when frequencies somewhat less than 1 per second are used. When higher frequencies are used, the errors in amplitude and wave form increase with the frequency.

Measurements at very low frequencies are difficult to make. However, at frequencies between 1 and 1.5 per second, the difficulties are reduced somewhat and the errors are not serious.

It was found that at frequencies above 2 per second the subject and the bed do not move in unison, and the pattern recorded depends in part on the elastic properties of the tissues of the subject. This leads to serious errors at high frequencies.

Complete specifications are given for the construction of the bed used in this work.

AUTHORS.

Gregg, D. E., and Shipley, R. E.: Augmentation of Left Coronary Inflow With Elevation of Left Ventricular Pressure and Observations on the Mechanism for Increased Coronary Inflow With Increased Cardiac Lead. Am. J. Physiol. **142:** 44, 1944.

Left coronary inflow is found to increase significantly in the anesthetized open-chest dog, when the load upon the left ventricle is increased by constriction of the aorta (central to the coronary orifices). This flow response is similar to that obtained previously in the right coronary artery in the presence of pulmonary artery constriction.

Measurement of cardiac input and oxygen consumption in the presence of an augmented load on either right or left ventricle has demonstrated an increase in the work and metabolism of the respective ventricle. In consideration of the increased coronary inflow observed, the two ventricles have at their disposal a compensatory means by which their blood supply can, at least in part, be adjusted to their work and metabolic requirements. It is suggested that the coronary dilatation arises from an increased local production of metabolites and/or the creation of local relative anoxia due to increased O_2 utilization.

If the experiments are intentionally prolonged (for several hours) and the coronary artery is perfused at constant pressure, the coronary flow response ultimately becomes a sustained decrease. As observed under these conditions, and by

others using heart-lung and isolated heart preparations, the decrease in coronary inflow is regarded as an "abnormal" response to an increased cardiac load.

AUTHORS.

Saracoglu, K.: A Rare Case of Complete Heart Block. Brit. Heart J. 6: 93, 1944.

A case of complete heart block with a rare degree of auricular bradycardia is described. The ventricular rate was 40 and the auricular rate 39 a minute.

As the auricular rate was not influenced by atropine or adrenalin it was thought to be due to coronary disease and not to increased vagal tone.

AUTHOR.

Neubauer, C. Heart-Block in Diphtheria. Brit. J. Child. Dis. 40: 93, 1943.

The author describes a series of forty-four cases of heart block in children suffering from diphtheria, observed from the onset to either convalescence or death. Of the cases described, thirty presented a partial, and fourteen a complete, heart block.

The various clinical signs are described, and an abbreviated report for each case is given.

McCULLOCH.

Logue, R. B., and Hanson, J. F.: Heart Block. A Study of 100 Cases With Prolonged P-R Interval. Am. J. M. Sc. 207: 765, 1944.

A series of 100 cases of heart block with prolonged P-R intervals is reported. The study consists of a review of 6,732 electrocardiograms taken on 4,264 patients at the Lawson General Hospital. The etiological classification of block is given with a discussion of causative factors. The P-R intervals of the 100 cases are tabulated, as well as variations in age and pulse rates. The normal range of the P-R interval is discussed. The belief is expressed that an occasional person may have a prolonged conduction time which is normal for that person and is perhaps associated with an individual variation of vagal tone. Observations are given regarding the effects of atropine in heart block with an expression of the authors' beliefs, as well as those of other observers, concerning its use.

AUTHORS

Scherf, D., and Klotz, S. D.: Electrocardiographic Changes After Acute Loss of Blood. Ann. Int. Med. 20: 438, 1944.

Fifteen cases of profuse gastric hemorrhage are reported. All but one showed alterations of the T wave, and some of the S-T segment as well, if electrocardiograms were taken soon after the hemorrhage, and daily thereafter. Of three cases of hemorrhage from other sources, two presented similar electrocardiographic alterations.

The first change to appear was lowering of the T waves. More advanced alterations consisted of disappearance or inversion of these waves and depression of the S-T segment. Slightly lowered voltage of the QRS complex was occasionally observed.

The changes in the electrocardiogram vanished in two to nine days; they developed in the absence of shock and without severe anemia and were independent of the hemoglobin level.

The effect of phlebotomy on the electrocardiogram was studied in thirty-three cases. Alterations were observed eight times. Although they may develop after the removal of 450 c.c. of blood, they may not appear with a venesection of 1,000 c.c. The appearance of electrocardiographic changes after an acute blood loss

has particular importance in the differential diagnosis between internal hemorrhage with cardiac symptoms and the cardiac manifestations associated with coronary thrombosis and pulmonary embolism.

AUTHORS.

Gross, R. E.: Complete Surgical Division of the Patent Ductus Arteriosus. Surg., Gynec. & Obst. **78:** 36, 1944.

Ligation of a patent ductus arteriosus has produced satisfactory closure in most of the fourteen cases in which it was used as the only method of obliterating the shunt. Better results were obtained in another series of twenty-eight patients in whom the ductus was ligated and was also wrapped with cellophane to promote regional fibrosis. While this latter method has been an improvement over the earlier procedure, it has failed in a few instances to produce absolute stoppage of the fistula. A new technique is now presented for complete division of the ductus which insures prompt and complete closure of this vessel. It has been performed in fourteen patients, two of whom had a superimposed *Streptococcus viridans* and endocarditis. All the patients have recovered, and none has had any important complication from the operative undertaking.

AUTHOR.

Weber, M. L.: Perforation of the Interventricular Septum Following Infarction; Intravitam Diagnosis. Ann. Int. Med. **19:** 973, 1943.

A modern review of the literature and an additional case of perforated septum following cardiac infarction, diagnosed before death, are presented. Comments on the clinicopathologic syndrome are made.

AUTHOR.

Sodeman, W. A.: The Systolic Murmur. Am. J. M. Sc. **208:** 106, 1944.

Interest in the significance of the systolic murmurs is especially important at this time when the question of physical fitness of draftees for military service comes up frequently. The author reviews all the important recent literature and correlates it with the early development of the interpretation of these murmurs in cardiology. The article is of considerable importance, since the need for correct interpretation of this murmur arises frequently in civil life as well.

MCCULLOCH.

Barron, D. H.: The Changes in the Fetal Circulation at Birth. Physiol. Rev. **24:** 277, 1944.

A great deal of information has been accumulated in recent years by direct methods of study carried out by Bancroft on the oxygen content of fetal blood, and by Barclay, using radiopaque substances in the blood stream to follow by cinematograph the pattern of circulation and the movement of blood, especially in the fetus delivered by cesarean section. The author reviews these studies, together with those carried out by other workers. He also reviews the recent work explaining the closure of the ductus arteriosus, ductus venosus, and foramen ovale. There is at present no satisfactory explanation of the mechanism initiating the closure of the ductus arteriosus. The available evidence indicates that the ductus arteriosus and the venosus are closed functionally within a few minutes after birth by the action of sphincter muscles appropriately placed. The closure of the foramen ovale may take place abruptly or immediately after birth, but the forces responsible for its closure have not been described.

MCCULLOCH.

Coburn, A. F.: The Prevention of Respiratory Tract Bacterial Infections. J. A. M. A. **126:** 88, 1944.

The United States Navy is engaged in a long-term program for the control of streptococcic infections and their disabling sequelae. One component of this program involves mass prophylaxis with sulfadiazine. Prophylactic doses of this drug were given continuously to about 250,000 naval trainees between December, 1943 and April, 1944. This preliminary report deals with observations on only 30,000 men at three camps.

These observations indicate that the continuous ingestion of 1 Gm. of sulfadiazine daily is adequate (a) to check a well-advanced streptococcic epidemic, (b) to check a streptococcic outbreak at its onset, and (c) to protect 85 per cent of susceptible recruits from implantation with bacterial respiratory pathogens.

These observations also suggest that a continuous daily dose of 0.5 Gm. of sulfadiazine (affording a mean level of 1.4 mg. per 100 c.c. in the blood and perhaps 0.8 mg. per 100 c.c. in secretions of the respiratory tract) is almost 85 per cent effective in preventing implantation by *Streptococcus haemolyticus.*

The only untoward effect of mass sulfadiazine prophylaxis is the occurrence of evanescent rashes in 0.5 per cent and dangerous constitutional disturbances in 0.01 per cent.

Mass sulfadiazine prophylaxis is effective (a) in checking bacterial infections of the respiratory tract, (b) in preventing the development of disabling sequelae caused by these bacteria, and (c) in aiding the economy of a nation at war.

AUTHOR.

Cluxton, H. E., and Krause, L. A. M.: Acute Lupus Erythematosus Disseminatus. Ann. Int. Med. **19:** 843, 1943.

Four cases, two colored and two white females, are used to emphasize the clinical aspects and to illustrate the variability of the symptom-complex.

Acute lupus erythematosus disseminatus is a disease of unknown etiology associated with widespread visceral lesions predominantly involving the kidneys, lymph nodes, blood vessels, serous and endocardial surfaces, as well as the skin. There is a marked predilection for females in the second and third decades. The prognosis is grave and the average duration of the disease is approxmiately eighteen months. The clinical picture is variable; however, the skin lesions, leukopenia with secondary anemia, arthritis, prolonged fever, and signs of renal involvement are prominent features that should make the diagnosis possible. The avoidance of any form of actinic therapy, except perhaps the roentgen irradiation of the ovaries, and the danger of eradication of foci of infection during the acute phase cannot be overemphasized.

AUTHORS.

Lambeth, S. S.: Peripheral Circulatory Collapse in Toxemia of Pregnancy. Am. J. Obst. & Gynec. **47:** 402, 1944.

Nine patients with varying degrees of circulatory collapse have been discussed. Whole blood plasma apparently is effective therapy for peripheral circulatory collapse in toxemia of pregnancy if used in the early stages.

AUTHOR.

Mahorner, H.: Control of Pain in Posttraumatic and Other Vascular Disturbances. Ann. Surg. **119:** 432, 1944.

Sixty patients, with conditions requiring temporary or permanent interruption of the sympathetic nerves, have been admitted to my private services at Touro

Infirmary and the Baptist Hospital. Twenty-two operations for permanent section of the sympathetic nerves have been done in this group.

Pains influenced by interruption of sympathetic nerve function are varied. They occur in vascular disease with associated vasospasm and may be attributable not only to ischemia, but also directly to the vascular spasm. Vascular changes occur and persist after trauma, and pain associated with these may prolong disability unduly. These pains may be surprisingly relieved by paravertebral sympathetic nerve blocks permitting more active physiotherapy for mobilization of joints. Such repeated nerve blocks may often shorten a convalescence dramatically. Cases are presented which illustrate this.

Sympathectomy is indicated in all patients with thromboangiitis obliterans under the age of 50 years. The operation gives many times more increase in vascular reserve in patients with early Buerger's disease than in those affected with a late stage, for which it is too frequently reserved.

Raynaud's disease is not a fault of the sympathetic nervous system. Sympathectomy for it is frequently disappointing, and eventually may be discarded entirely. These and other phases of the role of the sympathetic nervous system in vascular diseases were discussed.

AUTHOR.

Dauber, D. V.: Spontaneous Arteriosclerosis in Chickens. Arch. Path. **38**: 46, 1944.

Spontaneous arteriosclerosis of the aorta develops in 45 per cent of commercial roosters and hens over 1 year of age. The incidence of macroscopic lesions is the same in both sexes. Hens alone show fatty lesions of the intima of the ascending aorta and arch, while both roosters and hens commonly have intimal lesions of the abdominal aorta.

Arteriosclerosis in the chicken resembles human arteriosclerosis. Arteriosclerosis occurring spontaneously resembles that produced by cholesterol feeding.

The chicken is a suitable animal for the experimental production of arteriosclerosis if used before the age of 6 months, when spontaneous arteriosclerosis begins to occur. It is, furthermore, a suitable animal for studies on the prevention of arteriosclerosis because of the high incidence of the spontaneous disease and the early age at which it begins.

AUTHOR.

Pearce, M. B.: Varicose Veins. Surgery **14**: 901, 1943.

One hundred forty-one cases of varicose veins are reported. All those involving the long, saphenous were treated by uniform ligation of all its branches and the saphenous itself at the fossa ovalis without injection of sclerosing fluid into the thigh, together with ligation and injection of the internal saphenous at the lower thigh.

It is believed that complete emptying of the veins by compression bandages of the ACE or rubber type is a distinct advantage in reducing the size of the thrombi and insures better diffusion of the sclerosing fluid into the most distal ramifications of the varicosities.

Five cubic centimeters of sodium morrhuate produce excellent thrombosis in varicosities of the lower leg, and this is the maximum amount used in any single vein.

It would appear that this technique yields a complete obliteration of all varicosities without untoward manifestations.

No recurrences have been noted but it is unquestionably too soon to estimate this phase of the subject. It is hoped that a supplementary report may be made in the future.

AUTHOR.

Lauson, H. D., Bradley, S. E., and Cournand, A.: The Renal Circulation in Shock. J. Clin. Investigation **23**: 381, 1944.

The changes in renal vascular dynamics resulting from peripheral circulatory failure have been investigated by means of the clearance methods in thirty-five human cases. The study was part of the comprehensive investigation of the circulation in shock carried on at Bellevue Hospital.

The following conclusions may be drawn:

The rate of glomerular filtration and effective plasma flow are reduced in practically every case of shock. The reduction is variable, but roughly parallels the degree of shock.

In most cases, the decrease is greater than can be accounted for solely on the basis of reduced arterial pressure, suggesting active vasoconstriction in the renal vessels. The relationship between the renal blood flow and general circulation has been expressed in terms of two calculated values: The renal fraction, which designates the approximate proportion of total blood flow (cardiac output) which circulates through the kidneys, and the effective renal vascular resistance, which indicates the relation between systemic blood pressure and the renal blood flow. The decrease in renal fraction usually observed reveals that a smaller proportion of the cardiac output flows through the kidneys, indicating that blood is shunted away from the kidneys during shock. The increase in renal resistance indicates that renal vasoconstriction is the mechanism responsible for this redistribution of the circulation.

In general, the lowest clearances were associated with lowest blood pH values, but several lines of evidence indicate that acidosis is not the primary cause of decreased renal circulation. On the contrary, renal ischemia probably augments the acidosis resulting from widespread tissue anoxia.

Sources of error and limitations inherent in the clearance methods as applied to the study of shock are discussed. It is concluded that the clearances give a reasonably accurate description of the status of the renal circulation in this condition.

Urine flow is uniformly reduced and, in extreme cases, total anuria occurs. In general, the degree of oliguria reflects the reduction in rate of glomerular filtration, which in turn is related to the reduction in renal blood flow.

From a limited experience, the impression has been gained that acute alcoholism complicating slight or moderate oligemia tends to result in a relatively generalized vasodilatation, which is reflected in the kidney by a lower filtration fraction and a renal blood flow larger than in nonalcoholics with similar levels of blood pressure.

The influence of blood or plasma transfusion upon the clearances has been studied. There is a tendency for the filtration fraction to increase, suggesting efferent arteriolar constriction. The filtration rate increases with the rise in arterial pressure, but the renal blood flow tends to remain low or fall to subnormal values after a temporary increase.

In spite of the approximate return to normal of blood pressure and cardiac output, the renal circulation has, in most of the cases which have been adquately studied, failed to improve proportionately during the period of emergency treatment. However, measurements repeated several weeks later revealed a normal filtration rate and effective renal blood flow in all the cases so studied.

This investigation confirms the hypothesis that the urinary findings in shock, namely, oliguria or anuria, and loss or impairment of concentrating power, are the result of decreased circulation through the kidneys.

Therapy of shock in relation to the kidney is briefly discussed.

AUTHORS.

Blalock, A., and Park, E. A.: The Surgical Treatment of Experimental Coarctation (Atresia) of the Aorta. Ann. Surg. **119**: 445, 1944.

These experiments were undertaken with the idea of devising a means for the surgical treatment of coarctation of the aorta. At a one-stage operation the thoracic aorta was divided, the two ends were closed, and the proximal end of the divided left subclavian artery was anastomosed to the side of the aorta beyond the point of its division. Some of the animals survived this procedure, and there was evidence of adequate blood flow to the posterior part of the body. The possible clinical application of these studies is discussed.

AUTHORS.

Cooke, W. T., and Taylor, A. B.: Treatment of Subacute Infective Endocarditis With Heparin and Chemotherapy. Brit. Heart J. **5**: 229, 1943.

Five patients were treated with combined chemotherapy and intravenous heparin. All died, and, in one, this was probably due to the heparin therapy.

Twenty patients (including the five treated with heparin) were studied under treatment with sulfonamide derivatives. Five were totally unresponsive; twelve were moderately controlled. Life was prolonged in some, but all eventually died. Three patients became apyrexial; one died after forty-five days of normal temperature, but, as there was no autopsy, neither the diagnosis nor the state of the lesion could be determined. A second died one year after the onset of his infection and eight months after the control of his pyrexia by sulfapyridine; autopsy showed healed lesions of infective endocarditis. The third is well and working at his occupation as a milk-roundsman at the present time, twelve months after his discharge from the hospital.

Intravenous heparin did not prove of value in our cases and, as others have found, should not be used in these cases.

Prolonged chemotherapy offers a chance of cure to a few patients, though the great majority will not be so benefited. The dangers of such prolonged therapy are small and should not weigh against the chances of a successful outcome.

AUTHORS

McMillan, R. L.: Ventricular Tachycardia as a Therapeutic Problem in Coronary Thrombosis. South. M. J. **36**: 800, 1943.

Ventricular tachycardia as a complication of severe myocardial disease has been presented from the standpoint of etiology, pathologic physiology, diagnosis, treatment, and prevention. Two cases with successful quinidine therapy have been presented.

AUTHOR.

Governale, S. L., and Rink, A. G.: Spontaneous Renal Apoplexy With Resuscitation After Cardiac Arrest. Brit. M. J. **2**: 43, 1944.

A case of spontaneous renal apoplexy due to malignant hypertensive cardiorenal disease has been presented. The patient's heart stopped during operation. Cardiac resuscitation was accomplished by cardiac massage, intracardiac injection of stimulants, and intraventricular infusion of blood and saline. Life was restored for a period of eighteen hours after the operation. Death ensued from secondary or recurrent right renal hemorrhage. Perusal of the literature does not disclose any previously reported case of intracardiac or intraventricular infusion.

AUTHORS.

Book Reviews

HYPERTENSION AND HYPERTENSIVE DISEASE: By William Goldring, M.D., Associate Professor of Medicine, New York University College of Medicine, and Herbert Chasis, M.D., Assistant Professor of Medicine, New York University College of Medicine. The Commonwealth Fund, New York, 1944, 241 pages, 53 illustrations, $3.50.

Throughout this book the authors subscribe to the idiopathic, "functional," nonrenal origin of so-called *essential* human hypertension and to the secondary development of the organic vascular disease (intrarenal included) so frequently found associated with this condition. It is a striking and interesting fact, however, that in their enumeration of the abnormal conditions usually associated with true (systolic and diastolic) hypertension, all but three of the eleven conditions mentioned usually involve some type of renal abnormality. Although the authors admit that most of these diseases of the kidney, because they are "primary," may be the initial cause of the hypertension which follows their development, yet they deny that the organic vascular disease of the kidney of essential hypertension is the initial cause of the elevated blood pressure, because they consider, without proof, that the intrarenal vascular disease is secondary to the hypertension. The frequent occurrence of vascular disease (indistinguishable from that observed in essential hypertension) in organs other than the kidney in nonhypertensive individuals, the much more frequent (almost invariable) occurrence of vascular disease in the kidney in cases of essential hypertension, the experimental production of hypertension by a method which involves only a change in renal hemodynamics that reproduces the renal hemodynamic disturbance of human essential hypertension, and, finally, the absence of development of any changes resembling arterio- or arteriolosclerosis in animals with long-standing, benign, experimental, renal hypertension, fail to convince the authors of the possible primary renal origin of human, essential hypertension. Indeed, as an expression of the opposite view, this book is excellent and valuable.

The authors come to the unequivocal conclusion that the origin of essential hypertension is unknown but not renal. They agree that the elevation of the blood pressure is due to increased peripheral vascular resistance, and conclude that the primary mechanism which induces this must be humoral. Although they have shown that the only significant primary changes observed in the earliest cases of human essential hypertension involve functional hemodynamic and excretory alterations of the kidney, they conclude that these changes are due to the spasm of efferent glomerular arterioles and are therefore secondary to the hypertensive process which is initiated by a hypothetical humoral mechanism not of renal origin. They evidently attach little importance to the absence of efferent glomerular arteriolar spasm and of renal hemodynamic and excretory disturbances in types of hypertension that are not of renal origin, such as experimental neurogenic (carotid sinus hypertension), and humoral (pheochromocytoma of the adrenal) human hypertension. In neither of these types of hypertension have afferent glomerular arteriolar constriction, with diminution in effective renal blood flow, reduction of maximum tubular excretory capacity, and increase in glomerular filtration fraction been demonstrated. This means the attribution of a selective reaction to the efferent glomerular arterioles, which is hardly justifiable.

It is not the intention of the reviewer here to discuss the validity of the interpretation of the special measurements of renal hemodynamics and of renal execretory function devised by Homer Smith and used in all the studies referred to in

this book. Suffice it to say that the authors themselves admit that other interpretations, of at least some of the data, are possible, and that dogmatic statements about their significance in any estimation of the state of renal excretory function and hemodynamics are not yet entirely justifiable. Although sophistical and philosophical interpretation of scientific data is not only permissible, but desirable, the dangers must not be overlooked, and are recognized by these authors.

The material presented in the body of the book and the details given in the appendix constitute an excellent summary of the important contributions of, and methods developed by, Homer W. Smith and his various collaborators, among whom are included the authors of this book. The chapters on the medical and surgical treatment of hypertension and on the management of hypertensive disease are excellent critical summaries of these topics and constitute important parts of this valuable contribution to the literature on hypertension.

HARRY GOLDBLATT.

PATHOLOGY AND THERAPY OF RHEUMATIC FEVER: By Leopold Lichtwitz, M.D., late Chief of the Medical Division of Montefiore Hospital and Clinical Professor of Medicine at Columbia University, with a foreword by William J. Maloney, M.D., Grune and Stratton, New York, 1944, 225 pages, 69 illustrations, $4.75.

If this small volume were less excellent, it is conceivable that one might wish to temper criticism because of the untimely death of the author before its publication. But it stands so firmly on its merits, it contains so many admirable chapters, that there is no occasion for anything other than frank appraisal, so far as present knowledge and the limitations of reviewers will permit this.

It is perhaps the definitive statement of the theory which holds that allergic phenomena are the basis of rheumatic fever. Surely it is the most persuasive and eloquent presentation of this belief that has yet appeared, and it is difficult to believe that the evidence supporting it could be marshalled with greater skill or stated more lucidly than the author has done in this, his last work. The trend of thought in recent years, at least in this country, has been away from this hypothesis, but, until a specific agent has been proved solely responsible for all the manifestations of rheumatic fever, there will doubtless be many who will continue to regard allergy as playing some part in its genesis. Let it not be thought that the author has presented only the European point of view of fifteen years ago; he has indeed done this with great skill, but he has also considered all recent work bearing on the pathogenesis and treatment of the disease, including many papers published within the past two years.

To present the author's views fairly and adequately in his own words would require extensive quotations, but one can scarcely resist the temptation to extract a few words from the first two pages, which are devoted to a definition of rheumatic fever. "Rheumatic fever is a noninfectious disease. It is not caused by a specific microorganism or virus, but by a sensitization to antigens, protein in nature, which in most cases are products of microorganisms. These microorganisms may be either pathogenic or nonpathogenic. . . . Commonly, the sensitization tends to persist indefinitely, and in this chronic stage it may be either continuously or intermittently evident. When the sensitization rises, the rheumatic manifestations increase; when the sensitization falls below the threshold of clinical activity, the disease has an intermission. Reactivation is brought about not only by the specific antigen which initiated the disease, but also by a number of nonspecific factors, any one of which will affect the morbidly sensitized defense mechanism of the patient in a characteristically rheumatic manner. Thus fatigue, a chill, an adventitious infection, a slight injury, a touch of indigestion, a mental upset, or increased bodily activity may cause the rheumatic to relapse from a quiescent into an acute febrile state. . . . The rheumatic attack strikes at tissues

of mesenchymal origin. As these are an integral part of all organs or structures, local lesions may appear in any part of the body. . . . No matter where the rheumatic lesion occurs—whether in the heart, the meninges, or the joints—fundamentally, its nature is the same. It starts with a swelling of the base substance of the connective tissue. This swelling may clear up, or the process may go on to produce degenerative changes.''

There are splendid chapters upon the incidence of rheumatic fever and the influence of personal factors, upon its general pathology and systemic phenomena, and upon rheumatic disease of the heart and the blood vessels. Full and detailed consideration is given to the effects of the disease upon the joints, muscles, skin, nervous system, and various special organs or systems other than the cardiovascular. The longest chapter in the book, and one to be warmly commended to all interested in the clinical aspect of rheumatic fever, is that dealing with therapy. It is comprehensive, detailed, up to date, and informed by sound clinical judgment.

Especially to be commended is the arrangement of the extensive bibliography; every chapter is followed by a long list of appropriate references.

One cannot dismiss this volume without a word of heartfelt praise for the eight-page foreword by Dr. Maloney, which is a masterly summary of the book; a brief, but eminently fair, presentation of the views of the author. The opening page, relating to the discovery of salicylates, is one of the most charming and delightful accounts to be found in recent medical literature. There must be few, indeed, who can read this page and not continue eagerly through those that follow, revealing as they do the warm affection and admiration of Dr. Maloney for the author and for ''this great book.''

H. M. Marvin.

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